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THE

**PRACTICE OF PEDIATRICS**

IN ORIGINAL CONTRIBUTIONS

BY

**AMERICAN AND ENGLISH AUTHORS**

EDITED BY

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## PREFACE.

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A COMPREHENSIVE and authoritative survey of each of the major divisions of medicine is necessary from time to time to record its latest development and to enable those who desire to master it as a whole, or to post themselves on special points, to do so with facility. With this object three companion volumes have been arranged covering respectively Gynecology, Obstetrics, and Pediatrics, and furnishing a compact presentation of the world's best knowledge upon these closely connected departments.

The volume on Pediatrics, now in the reader's hands, is from the pens of well-known authorities in America and England, who have been selected as eminently fitted to write on the subjects assigned to them. These authors have kept in mind: first, the clinical picture of a disease, and second, the best methods for its treatment. This plan has allowed each author to give his own observations of a disease, and the therapeutic measures which have resulted in the greatest success. Naturally this adds to each contribution a personal element which is entitled to consideration, as the authors are, without exception, clinicians and teachers of wide experience.

In the arrangement of the volume more space than usual has been allotted to infant feeding, diseases of the alimentary tract, disorders of nutrition, respiration, and circulation, and to contagious diseases, the object being to describe the conditions most intimately associated with disease in children and not those which are more common in adult life and found but rarely in childhood. In a word, the line between Pediatrics and General Medicine has been carefully drawn, so that space has thereby been found for a full presentation of this specialty in a convenient volume. In some sections extra space has been given to methods of diagnosis which are now regarded as essential by physicians who wish to be exact in their work, but the details of which are not readily accessible elsewhere. On the other hand, mooted pathological questions have been omitted, and the pathology stated by each

(v)

author is limited to what is regarded as essential for a comprehensive knowledge of the disease with which it is associated.

The Editor's thanks are due to the authors for their contributions and for the care they have taken in revising their articles. Thanks are also due to Dr. Martha Wollstein, Pathologist to the Babies' Hospital, New York, and to Dr. David Bovaird, Jr., one of the contributors, for their valuable aid. To the Publishers, who have co-operated in making the volume attractive in every way, the Editor wishes to express his appreciation for the many courtesies they have extended.

NEW YORK, 1906.

W. L. C.

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# SECTION I.

## DISEASES AND INJURIES OF THE NEWBORN.

By EDWARD P. DAVIS, M.D.

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### CHAPTER I.

#### THE NORMAL INFANT—THE PREMATURE INFANT.

##### THE NORMAL INFANT.

IN order to understand the normal infant so as to appreciate pathological conditions it may be well briefly to consider the characteristics of the viable infant at full term.

**Size and Weight.**—Various criteria of viability have been considered important. The length of the finger-nails, growth of hair on the head, brightness and clearness of the eyes, weight, and the ability of the infant to nurse and to cry have been regarded as affording an accurate basis for the recognition of viability. Infants, however, differ so much in development that some more accurate data than these must be obtained for scientific judgment.

Dührssen's table, giving the length of the fetus at different periods of gestation, has been commonly accepted as practically accurate. By this we find that at six months the fetus is 30 cm. ( $11\frac{1}{8}$  in.) long, at seven months 35 cm. ( $13\frac{3}{8}$  in.), at eight months 40 cm. ( $15\frac{3}{8}$  in.), at nine months 45 cm. ( $17\frac{3}{8}$  in.), and at ten months 50 cm. ( $19\frac{3}{8}$  in.) long. The earliest recorded period of viability is twenty-six weeks, and at this time the fetus of average development should be 32 cm. ( $12\frac{1}{2}$  in.) in length. The difference between the length of the viable fetus and the length of the fetus at full term is sufficiently great to show that many infants may be born viable and yet sometime removed from full development.

In estimating the degree of development of the newborn infant we may have reference to the proportionate length, chest circumference, and cranial circumference. Thus, if the length be 50 cm. ( $19\frac{3}{8}$  in.) the circumference of the chest is half this plus 10 cm., or 35 cm. ( $13\frac{3}{8}$  in.), and the circumference of the cranium is 2 or 3 cm. greater, or 37 or 38 cm. ( $14\frac{1}{2}$  to  $14\frac{9}{16}$  in.). Essential variations from these proportions indicate lack of development and in some cases disease.

The average weight of full-term male infants 50 cm. ( $19\frac{3}{8}$  in.) long is 3274.10 grams; of female infants 49 cm. ( $19\frac{1}{8}$  in.) long, 3112.86 grams. As a general criterion of development Jung<sup>1</sup> observed that in well-developed or full-term infants the circumference of the shoulders equalled or exceeded the occipitofrontal diameter of the head.

In considering the characteristics of an infant at full term, Herz<sup>2</sup> found, in fully developed infants, lanugo widely developed over the entire body. He also observed that comedones were present upon the face in but 5.7 per cent. of full-term infants outside the region of the nose and lips, while in premature infants they extended over the entire face in 83.3 per cent. The skin of a normal infant is reddened and covered in many parts by vernix caseosa. After removal of the vernix caseosa the skin gradually becomes more pinkish in color. In premature infants the mammary glands are much less developed than in full-term infants, secretion forming much later or not at all.

**Umbilicus.**—The portion of umbilical cord remaining attached to the newborn infant is its only visible remnant of intrauterine existence, and this undergoes necrosis and separates from the infant between the sixth and eighth days of life. While the umbilical cord is free from bacteria at birth, numerous micro-organisms make their appearance in the stump within five or six hours. Among the pathogenic bacteria present are the staphylococcus pyogenes albus, citreus, and aureus. The number of bacteria is less in infants who are not bathed daily than in those who are. In view of such observations it becomes evident that the closure of the umbilical vessels must play an important part in the prevention of infection with these bacteria.

**The Blood.**—The blood of the newborn infant presents several characteristic features: the number of red cells is 6,000,000 to 7,000,000 per cubic millimetre, of leukocytes about 18,000; the hemoglobin per cent. is always above 100 and may reach 120; the specific gravity is 1060; nucleated red corpuscles are present in the proportion of  $\frac{1}{20}$  to  $\frac{1}{8}$  of the total number of leukocytes, and, finally, the hemolytic, bactericidal, and agglutinating power of the infant's blood serum is far less marked than in later life.

Within two weeks the red cells diminish from 5,000,000 to 4,500,000 per cubic millimetre, the leukocytes to 10,000, and the nucleated red cells also diminish in numbers. The lymphocytes number three-fourths to two-thirds of the total leukocytes.

Scipiades<sup>3</sup> found that both red and white cells diminish during the first ten days and this is equally true whether infants are bathed or not bathed. There is, however, after the initial loss of weight a greater gain in blood cells in bathed infants than in those who are not bathed. Late ligation of the cord does not prevent the early loss of cells, but in the long run it increases the volume of the fetal blood, and hence is indicated.

<sup>1</sup> Inaugural Dissertation, Bern, 1902.

<sup>2</sup> Klin. Untersuchungen an 100 Neugeborenen, Inaug. Diss., Freiburg.

<sup>3</sup> Archiv f. Gynäk., 1903, Band lxx.

**Circulation.**—Immediately after birth the heart beat does not differ materially from that which was heard within the womb. The impulse of the heart may be plainly felt by placing the finger-tips over the precordium. The reddish color of the baby's skin shows that oxygenation is going on and that asphyxia is absent. The pulse of a newly born infant varies from the first minute of birth. It may fall 20 or 30 beats and then be accelerated beyond the fetal rate. Usually it falls 10 to 30 beats, but the rate is easily increased.

As Ballantyne remarks, the physiological transition from the fetal to the postnatal form of circulation is no doubt very rapid, but the anatomical transition, evidenced by the obliteration of the lumina of the ductus arteriosus, foramen ovale, umbilical vessels, and ductus venosus may not be complete for some days or even weeks.

**Respiration.**—Respiratory movements of the newborn are at first abdominal and become thoracic only as the lungs expand. It is a question whether the use of the abdominal binder, by impeding the movements of the abdominal muscles, stimulates or retards the full development of respiration. The healthy full-term infant, so soon as its nostrils are freed from mucus, can breathe with the mouth shut and frequently does so. Persistent failure on the part of the infant to close the mouth during respiration indicates some abnormality in the nose or throat. The respiratory rate of the newborn is relatively high—from 30 to 45 per minute—but as the lungs expand it gradually falls. The condition known as atelectasis may, in premature and weak infants, be the cause of delayed aeration. The first cry of the infant plays an important part in expanding the lungs.

The full-term infant is able to suck vigorously. The fact that the infant can close its mouth on the nipple and keep the mouth closed for some moments shows that no pathological condition of moment exists in the nose or throat. When the baby drops the nipple to cough or becomes restless and disturbed during the effort to nurse, abnormality or disease in the respiratory tract should be suspected, and a proper examination made.

**The Temperature.**—The temperature of the fetus within the uterus has been ascertained by measurement to be above 100° F. The average temperature of the newborn is 99.5° to 100.2° F. There is a daily fluctuation from one-tenth to three-tenths of a degree. The temperature of the newborn falls after the first bath, and it must vary greatly in accordance with the precautions taken or the lack of care in preventing exposure to cold. It is not definitely known how low the temperature of the newborn can fall and not occasion death, but in my observation a newborn child was exposed on a winter's night for several hours, and survived.

**Kidney Action.**—Ferroni<sup>1</sup> examined the urine of ninety-two newborn infants during the first week of life, and believes that the character of this urine, the anatomical arrangement of the kidneys and the mechanical

<sup>1</sup> *Annali di Ost. e Gìn.*, 1902, p. 75.

conditions in the circulation of the newborn show that a true kidney function could not have been present in intrauterine life. A genuine secretion of urine does not take place until the third day. After the fourth day renal function is established. Nevertheless, Ballantyne believes that there is no room left for doubt that the fetal kidneys are at least occasionally active during fetal life. Albumin, casts, and uric acid are frequently present, and, in a few cases, sugar may be detected. The specific gravity of the urine varies between 1004 and 1010. When the specific gravity is lower the urine is less acid and the albumin and casts disappear. In ten cases Ferroni examined the urine of the newborn by cryoscopy. The results gave a proportion of 1.57. The urine of six newborn infants was studied to ascertain its toxic effects. This was found to be greater than that of the urine of older nursing children or of partly grown children or adults. The greatest degree of toxicity was present on the third or fourth day.

**Stomach and Intestines.**—The stomach is comparatively small and is more vertical than in the adult. Rotch (*Pediatrics*) shows a stomach with a capacity of 25 c.c. ( $\frac{2}{3}$  oz.), while the infant weighed 2500 gms. (5½ lbs.). The pylorus is situated immediately in front of the first lumbar vertebra. Anteriorly the stomach is in contact with the left under surface of the liver, while posteriorly it lies against the anterior surface of the spleen. The relations indicate, in part, the difference in its position in early infancy from those described in childhood. The greater curvature is over the transverse colon, which is often not definitely transverse. The whole intestinal canal is loosely attached to the posterior wall of the abdomen. The large intestine is more freely movable than the small. The appendix at birth usually measures from 3 to 5 cms. (1 to 2 in.) in length.

The newborn baby gives evidence of the condition of the intestines by the passage of meconium. This is made up of swallowed liquor amnii, lanugo hairs, vernix caseosa, epithelial cells from the skin and intestinal mucosa, bile, mucus, succus entericus, and pancreatic secretion. The absence of bile indicates obstruction of the bile-ducts. Microscopically, blood corpuscles and crystals of bilirubin and biliverdin are found.

**Weight of Organs.**—Legou<sup>1</sup> has investigated the proportionate weight of the various organs of the newborn. The upper extremities are in the same relation to the body as in the adult, but the lower extremities are much less developed. The heart weighs relatively one-fourth more than in the adult; the liver is one-third larger. The proportion between the size of the spleen and that of the remainder of the body in the fetus at term is the same as in the adult. The kidneys are one-third larger than in the adult, and at seven months' gestation the suprarenal capsules are proportionally fifteen times greater than in the adult individual. From the sixth month on the brain of the fetus is larger in proportion than that of the adult. The thymus gland is relatively large and varies

<sup>1</sup> Thèse de Paris, 1906, No. 179.

in weight from 8 to 13 gms. (2 to  $3\frac{1}{2}$  dr.). its relationship to the body weight is 1 to 250 to 1 to 350.

**Influence of Nursing.**—The act of sucking calls for increased respiratory effort and so furthers the unfolding of the lungs, while the movement of sucking assists indirectly in promoting the establishment of the circulation. While we recognize nursing as an important agent in promoting the interest of the mother, we must not forget that aside from the question of nutrition nursing has value in establishing the essential functions of respiration and circulation in the infant.

**Care at Birth.**—The change in the infant's surroundings which birth produces must under the best conditions be very great. Care should be taken that the room be suitably warm, the infant wrapped in a warm blanket and given artificial heat. While the hot-water bag is ordinarily sufficient it must be remembered that if the infant's body be moist or the bag leaks a severe burn may result. It is safer to surround the newborn infant with warmth which is perfectly dry.

As the blood of the newborn is excessive in hemoglobin it has no immediate need of respiration, after the act is once established, to maintain oxygenation. Hence, the head may be covered with a wrap of light flannel extending over the greater portion of the head and face.

The aseptic care of the cord is a matter of immediate importance, and is accomplished by ligating it firmly and by wrapping the stump in sterile gauze. The vessels of the cord sometimes slip within the ligature and secondary hemorrhage may result. An additional ligature may be required. Care should be taken in bathing the baby that neither bath sponges nor bath water come in contact with the cord, as sterility is maintained for a longer period when it is kept dry and allowed to mummify.

The eyes must be flushed with boric acid solution. A 2 per cent. silver nitrate or argyrol from 1 to 10 per cent. may be used if there is any question of gonorrheal infection.

The mouth may be washed with boric acid solution. That respiration may be unimpeded the nurse must see that the nostrils and mouth are free from mucus. The effort frequently made to remove mucus by inserting the finger wrapped in soft linen may fail and instead retained mucus may be carried farther into the respiratory passages. Both circulation and respiration will be aided, where these functions are slowly established, if the infant be held for a moment head downward and the trunk gently folded on itself anteriorly.

The secretion of urine will be facilitated by giving water freely, thus lessening the danger of uric acid infarctions and also one source of high temperature which is seen in infants who pass only a small amount of urine.

The common practice of oiling the newborn infant to further the removal of the vernix from its skin is advantageous if combined with gentle massage of the whole body. The first bath of the newborn, unless cautiously employed, may be a source of infection at the umbilicus, eyes, or mouth, or of such exposure as to bring about an attack of

pneumonia. A newborn infant should not be tubbed, but kept between the folds of a flannel apron and bathed by sponging or rubbing with absorbent gauze or cotton. Separate pieces of cotton or gauze should be used for the face and head and for other portions of the body. Bath-water should preferably be sterile, of moderate temperature, and the soap employed should be as pure as possible. If a newborn infant be rapidly and skilfully bathed, with light massage, the whole process conduces greatly to the stimulation of its vital functions. If this be badly done serious injury may be the consequence.

The healthy newborn infant gives abundant evidence of its normal condition. It cries but little, and only when disturbed. Its sucking or grunting sound gives evidence of its physical contentment. Its roseate color and the warmth of its body show a good circulation. Its power to swallow and to suck and its vigorous cry denote its strength. Its disposition to sleep when undisturbed gives evidence that its nervous system is not harassed by pain, cold, or other discomfort.

### THE PREMATURE INFANT.

From viability at twenty-six weeks to full term an infant is said to be premature. As the premature infant is less vigorous than the full-term infant, it sustains birth pressure less perfectly. In premature labor the membranes rupture before dilatation is complete and infection is more likely to occur. Abnormal presentations expose these infants to the added manipulation necessary to effect delivery. Many of the causes which terminate pregnancy before full term in the mother are conditions which render the infant feeble. Those diseases which produce wasting and weakness—*e. g.*, tuberculosis and syphilis—in the mother naturally weaken the child. Acute infections, such as typhoid fever, which attacking the mother bring on labor, also affect the infant. Premature separation of the placenta with intrauterine hemorrhage must necessarily depress the infant. The causes enumerated are sufficient, aside from the prematurity of the infant, to render it more feeble than normal and to make a prognosis of its survival guarded.

Premature infants are more susceptible to the depressing influence of cold, and also to infection than are full-term infants. As Ballantyne has pointed out, the circulation of premature infants is unsatisfactory because it is partly fetal and partly neonatal, the foramen ovale and the ductus arteriosus tending to remain patent longer than is normal in infants born at term, and in consequence the two blood currents are incompletely separated.

The conditions which predispose the newborn infant to infection have been most fully stated by Fischl.<sup>1</sup> Foremost among them is the fact that phagocytosis is much less marked than in later life, owing to the undeveloped condition of the lymph nodes, spleen, and bone-

<sup>1</sup> *Traité des mal. de l'Enf.*, par Grancher and Comby, T. v. p. 27.



marrow. The desquamation of the epithelial cells covering the skin and mucous membranes, as first noted by Epstein, decreases the power of these organs to resist the entrance of bacteria, and to this the incomplete development of the corneous layer of the skin (Hulot) also contributes. The protective power of the blood is much less marked than in adults; and, finally, the closure of the umbilical vessels is apt to be incomplete, and thus an entrance point for bacteria is present.

Premature infants are also very susceptible to drugs, and the quantity which would be safely borne by a full-term infant may destroy life. Observers have noted the extreme susceptibility of premature infants to poisoning with bichloride of mercury or carbolic acid when used as a wash.

It is not the absence of weight of the premature infant alone which determines its vigor or its possible survival. Some of the smallest premature children have developed best. Thus, Jardine<sup>1</sup> reports the survival of an infant born prematurely weighing two pounds. Shepherd saw a similar case. Mansell<sup>2</sup> reports the survival of a premature infant weighing at birth eighteen ounces, and in my own observation twins were born prematurely whose mother had been weakened by pneumonia. The boy weighed a little more than three pounds, the girl about two and three-quarters. These children have lived to be ten years old, and are vigorous and well developed.

Adrian<sup>3</sup> ascribes the weakness of premature infants to deficient production of heat and the fact that the functions of the lungs are poorly performed. In forty premature infants under his observation twenty-four died. Thirteen of these had some accident or disease which could be referred to the prematurity of the infants alone.

In deciding if the infant is premature we must not rely exclusively upon the weight, the appearance of the nails or hair, or other superficial criteria. The length of the infant is far more reliable as a basis of judgment. A viable and premature infant will be at least 31 or 32 cm. in length. Other criteria must be in keeping with this abnormal lack of development in length.

**Treatment.**—The treatment of these infants must begin with the conduct of premature labor by the obstetrician, who must exercise care to prevent pressure and infection.

Premature infants are very susceptible to the change in temperature which follows birth. The rectal temperature of a fetus in the uterus is 0.2 degree higher than that of the uterus itself. Under the most favorable circumstances the premature infant at birth is exposed to a change of temperature of 20° F.

To avoid chill a warm blanket should, if possible, be thrown over the infant so soon as it is expelled and even before the cord has been ligated. If the infant is born in breech presentation the trunk and lower extremities should be wrapped in warm sterile flannel or other

<sup>1</sup> British Medical Journal, 1902, vol. i. p. 654.

<sup>2</sup> Ibid., 1902, vol. i. p. 773.

<sup>3</sup> American Journal of the Medical Sciences, 1901, vol. cxxi. p. 410.

warm and sterile material (Fig. 1). Immediately after birth the premature infant should be placed in a warm receptacle and artificial heat placed about it. Especial care is necessary to protect the premature infant against draughts of cold air. A premature infant should not be bathed immediately after birth, as in the ordinary bath exposure is inevitable, and these infants do not resist infection so well as infants born at term.

Physicians and nurses should avoid handling such infants with cold hands. It will not do to trust to sensations in estimating the temperature of water or the air of the room, but the thermometer should be constantly employed.

FIG. 1



Incubator, showing infant bandaged with cotton.

**Dress.**—In dressing these infants the first care of the physician must be that the dress be thick, warm, comfortable, quickly applied, and easily changed. A warm blanket should be used over the infant before the cord is cut. The infant may be gently but thoroughly cleaned beneath a warm blanket by wiping and gently rubbing the skin with sterile cotton anointed with sterile olive oil. A dressing of sterile gauze or cotton may be retained upon the stump of the umbilical cord by a flannel abdominal binder.

I prefer to have the broad abdominal binder applied and then dress the infant in a loose gown or sack of flannel, without sleeves, which fastens about the neck and which is gathered below the limbs like a bag (Fig. 2).

In such clothing the infant can move freely without exposure to the air. Absorbent cotton and sterile gauze may be placed over the orifice of the urethra and over the anus to receive the discharges of urine and feces. At the time of birth it is well to flush the eyes gently with sterile water or dilute solution of boric acid. Credé solution of silver nitrate, 2 per cent., should be used in maternity hospital cases. The mouth may be gently but thoroughly cleansed with the softest linen dipped in 4 per cent. boric acid solution. The infant should be given a teaspoonful of warm water as often as it will take it.

**Incubators.** A very important question in the care of these infants is the selection of a suitable receptacle. This must be so arranged that a

FIG 2



Incubator, showing infant dressed in flannel sleeping bag.

fairly constant artificial heat can be maintained. For the first twenty-four hours, 95° F. are desirable. Unless the infant shows evidence of depression from this temperature it may be continued for several days. The temperature may gradually be lessened until 80° F. are reached. It is usual when possible to place premature infants in incubators. These vary in elaboration. As with other medical appliances, the simplest are most satisfactory. Ingenious and complicated incubators have been devised, many of which have apparatus for supplying oxygen to the infant in addition to heat. The disadvantage of elaborate incubators lies in their tendency to be infected on the inner surface. Those which are heated by gas do not furnish a favorable atmosphere for the infant. Such incubators are extensively advertised but are of little practical value.

If a permanent incubator be desired I have had excellent results with the use of Auvard's simple one (Fig. 3). This consists of a cubical box, across three-fourths of which extends a berth. There is free communication between the air chamber beneath the berth and the berth itself by the open space left where the floor of the berth does not meet the opposite wall. Beneath the berth are placed copper cans filled with hot water. I have found that if one of these cans be changed each hour a temperature of over 90° F. is readily maintained. A small trap-

FIG. 3



Incubator showing hot-water cans and arrangement.

door in the side of the box farthest from the opening into the berth permits air to enter, pass over the copper cans, and rise to the child lying in the berth. At the opposite extremity from the space where warm air arises a copper tube an inch or more in diameter is passed through the roof of the box. This tube may contain a small revolving fan kept in motion by the constant stream of heated air which finds egress through the tube. As the fan is delicate and likely to break down I have discarded it, using the simple tube alone. A constant circulation



of air is thus maintained in the simplest manner possible. The roof of the box next the tube is largely of glass, beneath which a thermometer is fastened. The box should not be placed upon the floor of the room, but upon a table or two chairs, and if desired it may be placed near a window or ventilator communicating with the outside air to secure the best possible air for the infant.

If a simple incubator be not available an excellent substitute can be improvised by taking the ordinary wicker clothes-basket in which clean linen is commonly placed (Fig. 4). Several cans filled with hot water or tightly corked bottles should be placed upon the bottom of the basket. A large warm blanket folded several times should line the floor and sides

FIG. 4



Clothes-basket prepared for use as an incubator.

of the basket above the bottles. A thermometer should be tied to the inside of the basket, so that it can be readily seen and measure the temperature of the interior of the basket. Additional blankets may be used to line the basket thoroughly and a blanket of medium weight should be placed over it. The infant should be placed upon the blanket in the basket above the cans or bottles. Sufficient space should be left in the covering to permit the free entrance of air. The basket should be placed upon a high table in a well-ventilated room and not crowded into a corner or over a register. A temperature of 90° F. or more may be readily maintained with this simple device by changing one or two of the bottles in the bottom of the basket at

regular intervals. If desired a hot-water bag covered with flannel may be placed in the basket next the infant. A basket so prepared is almost invariably available, is readily fitted, and answers every practical purpose.

While theoretically it would be desirable to supply the premature infant with oxygen, practically it is difficult to carry out and rarely necessary. With the basket incubator it would be difficult to introduce oxygen gas within the basket in sufficient quantity to benefit the child. Practically, if the air of the room be kept fresh and not above 70° F., and the blanket be not too closely applied, the infant will obtain oxygen sufficient. In mild weather the blanket covering the basket may be discarded if the infant does well.

**Stimulation.**—Premature infants require stimulation so soon as born. From two to five drops of the best brandy or whiskey, in two drachms of water, are sufficient. Other stimuli are not appropriate and seldom useful.

**Feeding.**—The problem of feeding the premature infant is especially difficult. The infant has at first not sufficient strength to nurse, nor can it be taken from its receptacle to the mother without danger. It is often so feeble that it cannot wait for the mother's milk to form, but must be fed within a few hours after birth.

Where the mother's milk is available and promptly and freely secreted it may be used by extracting it with a breast pump, keeping it at a suitable temperature—98° to 100° F.—and feeding it to the infant. This is done by partly filling a basin with hot water and placing in the basin a graduated glass already heated. The milk is then pumped from the mother's breast into the heated glass and taken immediately to the infant. Some prefer to discard the glass, placing the ball of the breast pump containing the milk in hot water so soon as the milk is extracted. It is well to thoroughly examine breast milk in these cases to be sure that it contains sufficient nourishment for the infant.

As a substitute for breast milk, the white of egg in water, whey, cows' milk, well diluted and predigested or modified, or chicken-broth are available. Albumen-water should be prepared with the white of an absolutely fresh egg in 8 ounces of boiled water, and a little salt may be added or it may be slightly sweetened with milk-sugar. Albumen-water may with advantage be combined with barley-water in many cases. In using cows' milk, if the infant be feeble, whey should be made or the diluted milk should be partially pancreatized. It will be remembered that at certain stages in this process the milk may become bitter and unpalatable. It is well to begin with a very moderate degree of heat, thus partially digesting the milk until the infant's powers of assimilation have been tested. If the infant for its age be sturdy the physician may decide to try milk which is not predigested. Then modified milk of low percentage should be employed, and such a formula as fat 1, sugar 7, proteid 0.50 is useful. Townsend's first formula<sup>1</sup> is as follows: Top milk from quart bottle, 1 ounce; water, 10 ounces; lime-water, 1 ounce; sugar of milk, 1 ounce.

<sup>1</sup> Archives of Pediatrics, 1894, vol. VIII.

This may be increased as needed. In some cases freshly made chicken-broth skimmed will be better digested than milk. It must not be forgotten that the premature infant requires a comparatively large quantity of water to flush the kidneys and intestines and to assist in starting the processes of digestion and assimilation.

The quantity of food and the intervals of feeding and stimulation are of great importance. A very feeble infant should be given not more than one drachm of food in the beginning at a feeding, as of Townsend formula. Food and stimulant should alternate, the infant receiving one of these every hour or hour and a half. As the premature infant has no idea of day or night, judgment is requisite in not disturbing it too frequently and yet in maintaining its nutrition (Figs. 5 and 6). If the infant's color be good and it is resting quietly it may usually go two or three hours at night without disturbance. It will soon form regular habits and thus learn to distinguish night from day.

To administer food to premature infants it is usually best to begin by dropping food and stimulant into the mouth with a pipette. An ordinary medicine dropper which has been thoroughly cleansed is convenient. A long pipette having a glass bulb graduated is very useful. Food and stimulant should be placed as far back as possible upon the infant's tongue so that the reflexes of the pharynx may be excited and deglutition result. As the infant gains in strength a small rubber nipple may be placed upon a small bottle and it may suck this nipple. The rubber bulb of a medicine dropper pierced with several needle holes often serves a useful purpose in this feeding. Townsend has used a glass tube with a nipple on one end and a rubber bulb on the other.

Few premature infants are so weak that they cannot be made to swallow by patient but gentle manipulation. Where such attempts are unsuccessful, food and stimulant may be introduced into the stomach by gavage. A small soft catheter (12 to 16 American scale), previously warmed, may be passed into the esophagus through the nostrils or directly through the mouth; a funnel being attached to this, the desired food and stimulant can be put into the stomach. In all cases where feeding is done by gavage it is essential that the infant should not be overfed; and as the amount of food given seems so small, the nurse often in her zeal overdoes it. The bowel of the premature infant is rarely available as a means of nutrition. The lower bowel is often partly filled with meconium, and absorption in these cases is less ready and successful than in older infants and children.

An auxiliary method of feeding a premature infant consists in the use of oils and fats by inunction. I have found by experience that the addition of alcohol or aromatic spirit of ammonia to oil renders it more readily absorbed; from 1 to 4 drachms of a mixture composed of 2 parts olive oil and 1 part alcohol can be introduced through the skin by gentle massage. Cod-liver oil would be especially valuable in some cases if its unpleasant and abiding odor did not make its use almost impossible. Inunctions with oil may be practised once or twice in

FIG. 5

DATE	TIME OF DAY	FOOD	TREATMENT	MEDICINE	WEIGHT	REMARKS	BOWELS	URINE
		ML.	BRAND	TYPE	GRM.			
April	1							
	2							
	3							
	4							
	5							
	6							
	7							
May	8							
	9							
	10							
	11							
	12							
	13							
	14							
June	15							
	16							
	17							
	18							
	19							
	20							
	21							
July	22							
	23							
	24							
	25							
	26							
	27							
	28							
August	29							
	30							
	31							
	1							
	2							
	3							
	4							
September	5							
	6							
	7							
	8							
	9							
	10							
	11							
October	12							
	13							
	14							
	15							
	16							
	17							
	18							
November	19							
	20							
	21							
	22							
	23							
	24							
	25							
December	26							
	27							
	28							
	29							
	30							
	31							
	1							

Chart of premature infant.



FIG. 6

DATE	TIME	TEMP.	PULSE	RESPIRATIONS	WEIGHT	LENGTH	HEAD CIRC.	CHEST CIRC.	ARM CIRC.	LEG CIRC.	FOOT CIRC.	BLOOD PRESS.	URINE	STOOLS	REMARKS
June 1	10:00	98.4	120	30	10.0	18.0	30.0	20.0	10.0	10.0	5.0	100/60	U	1	
2	11:00	98.6	125	32	10.2	18.2	30.2	20.2	10.2	10.2	5.2	100/60	U	1	
3	10:00	98.8	130	34	10.4	18.4	30.4	20.4	10.4	10.4	5.4	100/60	U	1	
4	11:00	99.0	135	36	10.6	18.6	30.6	20.6	10.6	10.6	5.6	100/60	U	1	
5	10:00	99.2	140	38	10.8	18.8	30.8	20.8	10.8	10.8	5.8	100/60	U	1	
6	11:00	99.4	145	40	11.0	19.0	31.0	21.0	11.0	11.0	6.0	100/60	U	1	
7	10:00	99.6	150	42	11.2	19.2	31.2	21.2	11.2	11.2	6.2	100/60	U	1	
8	11:00	99.8	155	44	11.4	19.4	31.4	21.4	11.4	11.4	6.4	100/60	U	1	
9	10:00	100.0	160	46	11.6	19.6	31.6	21.6	11.6	11.6	6.6	100/60	U	1	
10	11:00	100.2	165	48	11.8	19.8	31.8	21.8	11.8	11.8	6.8	100/60	U	1	
11	10:00	100.4	170	50	12.0	20.0	32.0	22.0	12.0	12.0	7.0	100/60	U	1	
12	11:00	100.6	175	52	12.2	20.2	32.2	22.2	12.2	12.2	7.2	100/60	U	1	
13	10:00	100.8	180	54	12.4	20.4	32.4	22.4	12.4	12.4	7.4	100/60	U	1	
14	11:00	101.0	185	56	12.6	20.6	32.6	22.6	12.6	12.6	7.6	100/60	U	1	
15	10:00	101.2	190	58	12.8	20.8	32.8	22.8	12.8	12.8	7.8	100/60	U	1	
16	11:00	101.4	195	60	13.0	21.0	33.0	23.0	13.0	13.0	8.0	100/60	U	1	
17	10:00	101.6	200	62	13.2	21.2	33.2	23.2	13.2	13.2	8.2	100/60	U	1	
18	11:00	101.8	205	64	13.4	21.4	33.4	23.4	13.4	13.4	8.4	100/60	U	1	
19	10:00	102.0	210	66	13.6	21.6	33.6	23.6	13.6	13.6	8.6	100/60	U	1	
20	11:00	102.2	215	68	13.8	21.8	33.8	23.8	13.8	13.8	8.8	100/60	U	1	
21	10:00	102.4	220	70	14.0	22.0	34.0	24.0	14.0	14.0	9.0	100/60	U	1	
22	11:00	102.6	225	72	14.2	22.2	34.2	24.2	14.2	14.2	9.2	100/60	U	1	
23	10:00	102.8	230	74	14.4	22.4	34.4	24.4	14.4	14.4	9.4	100/60	U	1	
24	11:00	103.0	235	76	14.6	22.6	34.6	24.6	14.6	14.6	9.6	100/60	U	1	
25	10:00	103.2	240	78	14.8	22.8	34.8	24.8	14.8	14.8	9.8	100/60	U	1	
26	11:00	103.4	245	80	15.0	23.0	35.0	25.0	15.0	15.0	10.0	100/60	U	1	
27	10:00	103.6	250	82	15.2	23.2	35.2	25.2	15.2	15.2	10.2	100/60	U	1	
28	11:00	103.8	255	84	15.4	23.4	35.4	25.4	15.4	15.4	10.4	100/60	U	1	
29	10:00	104.0	260	86	15.6	23.6	35.6	25.6	15.6	15.6	10.6	100/60	U	1	
30	11:00	104.2	265	88	15.8	23.8	35.8	25.8	15.8	15.8	10.8	100/60	U	1	
31	10:00	104.4	270	90	16.0	24.0	36.0	26.0	16.0	16.0	11.0	100/60	U	1	
July 1	11:00	104.6	275	92	16.2	24.2	36.2	26.2	16.2	16.2	11.2	100/60	U	1	
2	10:00	104.8	280	94	16.4	24.4	36.4	26.4	16.4	16.4	11.4	100/60	U	1	
3	11:00	105.0	285	96	16.6	24.6	36.6	26.6	16.6	16.6	11.6	100/60	U	1	
4	10:00	105.2	290	98	16.8	24.8	36.8	26.8	16.8	16.8	11.8	100/60	U	1	
5	11:00	105.4	295	100	17.0	25.0	37.0	27.0	17.0	17.0	12.0	100/60	U	1	
6	10:00	105.6	300	102	17.2	25.2	37.2	27.2	17.2	17.2	12.2	100/60	U	1	
7	11:00	105.8	305	104	17.4	25.4	37.4	27.4	17.4	17.4	12.4	100/60	U	1	
8	10:00	106.0	310	106	17.6	25.6	37.6	27.6	17.6	17.6	12.6	100/60	U	1	
9	11:00	106.2	315	108	17.8	25.8	37.8	27.8	17.8	17.8	12.8	100/60	U	1	
10	10:00	106.4	320	110	18.0	26.0	38.0	28.0	18.0	18.0	13.0	100/60	U	1	
11	11:00	106.6	325	112	18.2	26.2	38.2	28.2	18.2	18.2	13.2	100/60	U	1	
12	10:00	106.8	330	114	18.4	26.4	38.4	28.4	18.4	18.4	13.4	100/60	U	1	
13	11:00	107.0	335	116	18.6	26.6	38.6	28.6	18.6	18.6	13.6	100/60	U	1	
14	10:00	107.2	340	118	18.8	26.8	38.8	28.8	18.8	18.8	13.8	100/60	U	1	
15	11:00	107.4	345	120	19.0	27.0	39.0	29.0	19.0	19.0	14.0	100/60	U	1	
16	10:00	107.6	350	122	19.2	27.2	39.2	29.2	19.2	19.2	14.2	100/60	U	1	
17	11:00	107.8	355	124	19.4	27.4	39.4	29.4	19.4	19.4	14.4	100/60	U	1	
18	10:00	108.0	360	126	19.6	27.6	39.6	29.6	19.6	19.6	14.6	100/60	U	1	
19	11:00	108.2	365	128	19.8	27.8	39.8	29.8	19.8	19.8	14.8	100/60	U	1	
20	10:00	108.4	370	130	20.0	28.0	40.0	30.0	20.0	20.0	15.0	100/60	U	1	
21	11:00	108.6	375	132	20.2	28.2	40.2	30.2	20.2	20.2	15.2	100/60	U	1	
22	10:00	108.8	380	134	20.4	28.4	40.4	30.4	20.4	20.4	15.4	100/60	U	1	
23	11:00	109.0	385	136	20.6	28.6	40.6	30.6	20.6	20.6	15.6	100/60	U	1	
24	10:00	109.2	390	138	20.8	28.8	40.8	30.8	20.8	20.8	15.8	100/60	U	1	
25	11:00	109.4	395	140	21.0	29.0	41.0	31.0	21.0	21.0	16.0	100/60	U	1	
26	10:00	109.6	400	142	21.2	29.2	41.2	31.2	21.2	21.2	16.2	100/60	U	1	
27	11:00	109.8	405	144	21.4	29.4	41.4	31.4	21.4	21.4	16.4	100/60	U	1	
28	10:00	110.0	410	146	21.6	29.6	41.6	31.6	21.6	21.6	16.6	100/60	U	1	
29	11:00	110.2	415	148	21.8	29.8	41.8	31.8	21.8	21.8	16.8	100/60	U	1	
30	10:00	110.4	420	150	22.0	30.0	42.0	32.0	22.0	22.0	17.0	100/60	U	1	
31	11:00	110.6	425	152	22.2	30.2	42.2	32.2	22.2	22.2	17.2	100/60	U	1	

Chart showing gain of premature infants treated by incubation, artificial feeding, massage, and oil  
inunction at the Jefferson Maternity Hospital.

twenty-four hours and have the additional advantage that the massage which accompanies the inunctions stimulates the infant's circulation, and its assimilation. Such massage should be done beneath a flannel sack and without exposing the infant to the external air.

The care of the intestine in the premature infant is of great importance. Meconium is frequently retained and the development of digestion retarded through sluggish action of the intestine. I have found daily irrigation of the bowels with equal parts of normal salt solution and boiled water of especial value. This must be not less than 100° F., and should be given with a funnel and not with a piston or valve syringe. It should be done at a regular time when it is desirable to have the bowels move. Occasionally, it is necessary to do this twice in twenty-four hours, but care must be taken not to irritate the intestinal mucous membrane. In cases where the bowel becomes irritable salt solution may be replaced by two ounces of warm olive oil. This will encourage a movement of the bowels, and a little of the oil may be retained to advantage.

In the general care of premature infants, patience and good sense are of the greatest importance. The premature infant should not be removed from the incubator until it has attained the age of normal development and continues to gain in weight and vigor. With some mothers it is difficult to maintain a good secretion of milk without the stimulus of the infant's nursing. Besides the physical there is to some extent a psychical element in the presence of the infant, and when this is lacking the supply of milk may diminish or cease. Care should be taken to explain to the mother that the enforced absence of the infant will terminate as soon as possible, and she should be encouraged to hope that she will be enabled to nourish the infant until it can be applied directly to the breast.

As the infant begins to gain in weight its oil inunction may be accompanied by general massage combined with massage of the intestine, gently given for twenty minutes or half an hour. This develops the muscles, stimulates the circulation, and improves the infant's nutrition.

Absolute regularity should be observed in the care of premature infants. As they are not yet sufficiently developed to notice objects about them, this care is more easy than in the case of infants born at term. Premature infants in incubators properly cared for usually cry less than full-term infants, partly because they are disturbed so little and partly because they are weaker. To care for such an infant two nurses are necessary. For several weeks the infant must be constantly watched, and this is almost impossible with but one nurse, even though some friend or relative assists.

It is not infrequent for premature infants to lose slightly or remain stationary in development for a short time after birth. So long as the infant's strength is well maintained, its movements well digested, and it is not fretful this need occasion no alarm. After a slight pause it will usually commence to gain. If, however, the infant loses considerably or fails within a reasonable time to gain, then some essential change in

its hygiene must be made. In order to estimate the progress of such an infant it must be weighed at regular and frequent intervals. It is safest to weigh the incubator with its contents, and, knowing the weight of the incubator and appendages without the infant, the weight of the child is readily obtained. If this is impossible, then the infant must be placed upon the scales, every precaution being taken to avoid chill. A further means of estimating the development of a premature infant consists in ascertaining and recording its length. By reference to the chart (Fig. 6) it will be observed that a considerable increase in length accompanied the gradual growth of the infant. It is not infrequent for an infant while growing in length to remain stationary in weight. If this be known the failure of the infant to gain in weight is explained. If the infant does well it may leave the incubator permanently when it has come to full term, it being possible to secure for it adequate protection against cold.

The chance for a premature infant born in the spring or early autumn is somewhat better than that of a child born in winter or in the heat of midsummer. Premature infants are so sensitive that they feel extremes in temperature even though protected by an artificial environment. In early summer the lid of the incubator may be removed and the infant may be given sun baths at a temperature as nearly as possible that maintained by artificial heat.

**Prognosis.**—A physician will do wisely to withhold a prognosis regarding a premature infant. While many survive, others do not, and some fail without appreciable cause. The influence of infection must not be forgotten, as it may be the cause of death. Sudden death is not uncommon in these cases and nurses should be warned of this fact in undertaking their care. Death sometimes occurs in convulsions, but most often quietly and with so little disturbance that the death of the infant may not be recognized for some time. The state of the heart, the power of digestion, the action of the lungs, and the influence of infection all affect the prognosis.

## CHAPTER II.

### ASPHYXIA NEONATORUM—ACCIDENTS TO THE UMBILICAL CORD.

#### ASPHYXIA NEONATORUM.

By the term asphyxia we understand lack of oxygenation of the blood with consequent accumulation of carbon dioxide and its poisonous effects upon the nerve centres. Asphyxia may be intrauterine, the infant perishing before birth, or it may become apparent after the infant has been expelled from the uterus.

**Intrauterine Asphyxia.**—Disease or premature separation of the placenta, prolongation of the second stage of labor from any cause, or death of the mother may cause intrauterine asphyxia.

**Extrauterine Asphyxia.**—This form of asphyxia of the newborn commonly arises from occlusion of the umbilical cord through coiling or prolapse of the cord with pressure. Congenital atelectasis may be associated with asphyxia. It sometimes arises from the inspiration of mucus, amniotic liquid, or blood. It may also follow birth pressure, which may produce cerebral or pulmonary hemorrhage. Its effect on the future health of the infant may thus be most important.

**Symptomatology.**—Asphyxia has been divided into livid or blue asphyxia and pallid or pale asphyxia. In the livid or blue asphyxia the infant's color is dusky reddish-blue, the heart beat is evident, the muscles are not completely relaxed, the pupils are not widely dilated, and the reflexes are, to some extent, present. In pallid or pale asphyxia the infant's color is cadaveric white, its heart beat is imperceptible or very feeble, its pupils are widely dilated, and its reflexes cannot be excited.

**Treatment.**—The prevention of asphyxia is entirely obstetrical. Prolonged labor with excessive birth pressure and injury to the cord must be avoided. Late ligation of the umbilical cord indirectly helps to prevent asphyxia, as it gives to the infant a greater quantity of oxygenated blood, thus supporting its circulation.

In the treatment of *livid* or *blue* asphyxia it must be remembered that the infant resembles a clock which has been wound, but whose pendulum must be moved to put the works in motion. What is needed in these cases is to excite respiration by arousing the nervous reflexes. If the cord is beating and the physician allows pulsation to cease spontaneously before tying and cutting the cord, he should then determine the presence of fetal heart beats by auscultation or by pressing with the finger-tips against the apex of the heart. Where asphyxia is slight, slapping the infant lightly, dashing a few drops of cold water on

the chest, placing the infant in a warm bath, and spraying a little cold water upon the chest will arouse the muscles of respiration. If the infant seems plethoric and oppressed with blood it may be allowed to lose a few drachms of blood from the cord.

If the finger be dipped in whiskey and carried downward into the fauces the infant will make a sucking motion and may then respire.

Laborde's<sup>1</sup> method of making rhythmical traction upon the tongue is endorsed by Rivemont-Desaignes,<sup>2</sup> and also by Fronczak,<sup>3</sup> who believes that it is safer than those methods which expose the child to rapid cooling of its body and to the danger of injury to the clavicles. Laborde, in investigations made to determine the length of time after apparent death in which the reflexes could be excited, found this period to be three hours, and would continue rhythmical tractions upon the tongue for that length of time.

Cases of livid asphyxia require especial attention to the cutaneous reflexes. Gentle friction while the infant's body is immersed in a bath of warm water containing mustard acts as a powerful stimulant to respiratory reflexes. The external application of warmth is less necessary in these than in cases of pale asphyxia.

I believe that in cases of livid asphyxia the right heart of the infant and the large veins of the body are overdilated with blood. The simple manœuvre of folding and unfolding the body of the infant, proposed and described by various observers, I have found of great value. After the mouth has been thoroughly cleansed of mucus and the cord tied and cut, the infant is grasped with one hand across the back, the fingers resting upon the clavicles; the other hand grasps the thighs. Holding the infant with the head down, the trunk is then folded and unfolded. From ten to sixteen may be counted during each movement of the child's body. During folding the abdominal viscera are carried up against the diaphragm, the diaphragm is pushed upward, whatever air may be in the lungs is forced out, while the pressure brought to bear upon the abdominal viscera forces the blood upward from the abdominal veins and the pressure of the diaphragm against the heart and lungs tends to promote the emptying of the chambers of the heart. When the infant is unfolded air may enter the lungs, the pressure on the veins is removed, and the conditions are more favorable for the circulation of arterial blood. So successful in my experience has this simple manœuvre been in the treatment of asphyxia that it has largely superseded other methods of treatment. The fact that it enables us to directly stimulate the circulation by simple means while furthering the establishment of respiration makes the method especially valuable, even in the *treatment of pale asphyxia* where the problem is more difficult, for oxygen must be introduced into the blood and as rapidly as possible to remove the paralyzing effects of the carbon dioxide already accumulated. The physician must not only introduce air into

<sup>1</sup> *Gaz. des hôpitaux*, 1901, tome lxxiv. p. 1319.

<sup>2</sup> *Annal. de Gynéc.*, 1900, tome liv. p. 101.

<sup>3</sup> *Buffalo Medical Journal*, vol. iv. 1899, 1900.



the child's chest, but he must stimulate the action of the heart, maintain the warmth of the body, while avoiding injury to the child by any method of treatment.

To secure the entrance of air into the lungs artificial respiration may be practised. Marshall Hall's method and Sylvester's method have their advocates and have in some cases proven efficient.

FIG. 7



Schultze's method: Infant going downward for inspiration.

FIG. 8



Schultze's method: Infant over operator's shoulder for expiration.

Schultze<sup>1</sup> describes his method as follows: While the operator stands the infant is grasped with both hands, fingers resting over the scapulæ and the thumbs upon the anterior surface of the chest near the clavicles (Figs. 7 and 8). The infant's body is allowed to fall downward toward the floor with a swinging motion. In the same way it is then raised at arm's length over the operator's head and then with a long swing it is again brought downward toward the floor. By the upward motion the infant's body is bent upon itself, the abdominal viscera are crowded upward against the diaphragm, and expiration is promoted. With the

<sup>1</sup> Die Behandlung des Scheintodes der Neugeborenen, Wiener med. Presse, 1900, Bd. xli, p. 1518.

downward motion the abdominal viscera gravitate away from the diaphragm, a vacuum is created in the bronchial tubes, and air rushes into the lungs. Abundant postmortem examinations show that by this manipulation air can undoubtedly be forced into the lungs. Schultze is aware that this method exposes the child to considerable disturbance, and he advises Sylvester's method with Pazini's modification in prematurely born infants who are not of full strength and development. Schultze argues that other methods of treatment serve only to excite reflexes if such be present, while by his method air is actually introduced into the lungs. Fracture of the clavicle, as well as other injuries, has been observed, and it is also urged against this method that the child's body becomes rapidly cool and that its use is attended by considerable exposure.

The use of oxygen would theoretically meet the indications in pallid asphyxia. It is doubtful, however, whether oxygen can be introduced into the lungs without tracheotomy. Stowe<sup>1</sup> reports the case of a child, severely asphyxiated after birth in breech presentation, revived by tracheotomy, the introduction of a catheter, and insufflations of air.

Zangemeister<sup>2</sup> introduced oxygen through a small tracheal catheter under feeble but constant pressure. After the lungs had become distended he allowed thoracic pressure to expel the oxygen; the result was satisfactory.

One of the simplest and oldest methods for introducing air into the chest has been direct insufflation. A handkerchief was hastily thrown over the mouth of the infant and the physician, applying his mouth, breathed deeply into the infant's mouth. Then by gentle pressure upon the chest the air was expelled. This method exposes the infant to tuberculous and other infection from the mouth of the adult, while the thoracic pressure is not always without danger. Others have employed the tracheal catheter with the balloon, thus introducing air directly into the larger bronchial tubes. The direct insufflation of air is but partially satisfactory and is inferior to the introduction of oxygen by direct application through the catheter or after tracheotomy.

In treating cases of severe asphyxia the physician must keep in mind that he is dealing not only with failure of respiration, but that the infant is suffering from cardiac syncope. Hence, those methods should be employed which promote the action of the heart. Such are the use of external heat, counterirritation over the precordium, the use of the interrupted faradic current, one pole at the base of the brain, the other over the heart, and the injection of a half-teaspoonful of whiskey in a half-ounce of warm water into the rectum. I have seen good results follow the administration of 0.00021 grm. ( $\frac{1}{4000}$  gr.) of strychnine with 0.000014 grm. ( $\frac{1}{70000}$  gr.) of atropine by hypodermic injection. The finger may be dipped in whiskey and placed within the fauces as above described. The limbs of the infant may be gently rubbed from below

<sup>1</sup> *Gaz. hebdom. de méd. et de chir.*, 1902, p. 10.

<sup>2</sup> *Zentralblatt f. Gynäk.*, 1902, Bd. xxvi, p. 1161.

upward, and normal salt solution may be injected into the umbilical and into the rectum.

Although no reference is found to the use of adrenalin it would be reasonable to suppose that half a drachm of 1:10,000 solution might be introduced through the umbilical vein to advantage.

The resuscitation of an infant severely asphyxiated may be followed by complications. Thus inspiration pneumonia has resulted from drawing of infected material into the bronchial tubes. In cases of severe asphyxia complicated by birth pressure I have seen pulmonary apoplexy during the first twelve hours following delivery. Cerebral hemorrhage has been found in some cases of severe asphyxia. Infants were temporarily revived. From manipulation about the mouth and tongue, wounds and infection have resulted. Jacobi has observed epilepsy following resuscitation from severe syncope. Snow<sup>1</sup> reports two cases of failure of respiration with cyanosis of central origin. Cyanosis and respiratory failure have also been observed early in life. Dusting powders containing coal-tar derivatives have been used on the cord.

**Prognosis.**—In livid or blue asphyxia with intelligent treatment the prognosis is good. It is sometimes impossible to decide positively if an asphyxiated infant is beyond resuscitation. Infants apparently dead have been repeatedly thrown into various receptacles and have survived.

Redfern and Newby<sup>2</sup> describe a case of an asphyxiated infant whose heart continued to beat without respiration for two and a half hours after birth. Tracheotomy was performed and breathing finally instituted. Several hours after the infant had been made to breathe it perished through a secondary asphyxia.

In view of these remarkable resuscitations, efforts to revive asphyxiated infants should be patient and prolonged. So long as the faint evidence of heart beat exists, efforts to revive the infant must be continued. Especial attention must be directed to maintaining the heat of the infant and to avoiding all violent manipulation.

The mortality of asphyxia in the newborn cannot be accurately stated. Cases differ greatly in severity; the circumstances under which treatment is instituted vary greatly, and the presence of hidden complications which make the case hopeless is often not demonstrable at autopsy.

#### ACCIDENTS TO THE UMBILICAL CORD.

Rupture of the umbilical cord, either preceding or following birth, may destroy the life of the infant. Such an accident is not invariably fatal, because the vessels of the cord may retract and serious bleeding may thus be checked. A hematoma may form and hemorrhage gradually cease through pressure.

<sup>1</sup> Archives of Pediatrics, October, 1903.

<sup>2</sup> British Medical Journal, 1901, vol. II, p.



Bayer states that in 48 precipitate births rupture of the cord occurred in 7, or 14.5 per cent.

Among those conditions which predispose to rupture of the cord Bondi<sup>1</sup> calls attention to syphilis.

**Hemorrhage.**—Primary bleeding from the cord is due to violence or slipping of the ligature, an accident to which a large amount of Wharton's jelly predisposes by making compression of the vessels difficult.

Secondary bleeding (spontaneous or idiopathic omphalorrhagia), described by Runge as "not a disease, but a symptom of various morbid states," is a steady oozing and not a hemorrhage from any single bloodvessel. Hereditary hemophilia is rare in these cases; syphilis is apparent in some, but the majority of them are due to septic infection. Streptococci, staphylococci, and diplococci have been demonstrated at the umbilicus and in the blood of the patients.

The onset of the bleeding may be insidious and generally follows the separation of the umbilical stump, or the general symptoms of septic infection may precede it. By far the greater number prove rapidly fatal.

**Treatment.**—Under all circumstances the cord should be tied firmly and carefully. It is well to employ two ligatures, placing one a finger's breadth from the umbilicus and the other near the extremity of the stump. Pedicle silk, or silk a size larger, is safest, and fine silk may be used to tie the individual vessels if desired. All ligatures should be thoroughly sterilized by boiling. Catgut is an unsafe ligature for the umbilical cord because of the danger of slipping, its uncertain sterilization and the risk of infecting it while manipulating the cord.

The cut end of the cord should be sponged and examined carefully to detect oozing. The vessels should be seen to be empty and the cut end of the cord dry. The cord should be dressed aseptically in such a manner that it will be disturbed as little as possible and that traction upon the knots of the ligature may be avoided. So soon as possible a firm but not tight abdominal binder should be pinned about the abdomen, thus making pressure upon the umbilicus.

Where there has been rupture of the cord close to the abdominal wall, it may be necessary to transfix the stump by needles placed at right angles. Pressure may then be made with a figure-of-eight silk ligatures. If the cord is thick and there is a great quantity of Wharton's jelly, it is necessary to strip the cord before ligating it. Ruptured and soft cords may be irrigated with normal salt solution and then washed with alcohol before applying the ligature. The bleeding may sometimes be controlled by pressure applied with a pad or by forceps allowed to remain twenty-four hours or more. A compress may be soaked in a solution of adrenalin—1:1000—wrapped firmly about the cord and pressure applied. Occasionally it is possible to isolate some bleeding point, to seize this with the hemostatic forceps, and apply a ligature. Styptics are of little or no value either in the primary bleeding or in the later oozing.

<sup>1</sup> Zentralblatt f. Gynäk., 1903, Bd. lxi. p. 223.

**Septic Infection.**—Septic infection of the umbilical cord stump may be limited to the umbilical cicatrix only; or, if the infecting organism be the streptococcus, the inflammatory process may spread to the surrounding skin and cellular tissue through the lymph channels, and omphalitis or even erysipelas result. Extensive ulceration and gangrene may follow. General infection with or without thrombosis and inflammation of the vessel walls may occur if streptococci or staphylococci find entrance into the umbilical vein or arteries. The class of infections where there are no distinctive local signs frequently go undetected until constitutional symptoms supervene.

**Treatment.**—The enforcement of rigid asepsis in the case of the umbilicus is the best prophylaxis. Where sepsis is already evident local measures are indicated. Irrigation with normal salt or boric acid solution are useful. If corrosive sublimate solutions are employed they must be well diluted, as infants are susceptible to their toxic effects. Preparations of peroxide of hydrogen will search out pus in the interstices around the umbilical ring. Local abscesses are to be treated surgically. It is advised that these patients be kept face downward so as to allow of drainage. Stimulation by brandy or whiskey is necessary.

**Umbilical Fungus.**—Umbilical fungus or granuloma is a mass of granulations and indicates defective healing of the umbilicus. When the granulation tissue is touched it may bleed, and, as the skin around the umbilicus is kept moist by the discharge of serum or sero-pus there is often an annoying eczema.

**Treatment.**—After a careful cleaning with normal salt solution the mass may be ligated, or, if too small to hold a ligature, it may be then destroyed by one or two applications of the actual cautery.

## CHAPTER III.

### INJURY AT BIRTH—INFECTIONS.

#### BIRTH PRESSURE.

THE most important element in producing injury to the cranium and its contents during labor is the continuance of birth pressure, especially where there is a disproportion between the pelvis and the fetal head.

This pressure may cause such a well-known condition as a cephalhematoma, or it may be sufficient to produce a fracture of one or more of the cranial bones. The meninges may be torn, the brain lacerated, and extravasations of blood and cerebrospinal fluid may do fatal injury to important nerve centres. Lacerations of the ears, mouth, and soft tissues, with injuries to the eyes, may occur.

A study of the mechanism of labor teaches us that it is not the forceps properly applied, but delay in labor with continuous birth pressure which causes injury. The proper use of forceps undoubtedly prevents injury in many cases, and threatened danger to the fetus by continued pressure is a valid indication for delivery by forceps. Extreme effects of pressure are shown in Figs. 9 and 10.

#### CEPHALHEMATOMA.

Among the most common of the injuries received by the newborn infant is that of pressure upon the cranium, which results in the formation of cephalhematoma and also somewhat less frequently in injuries to the sternocleidomastoid muscle. Two factors combine in its causation: defective ossification of the cranial bones at the point where the hemorrhage develops and the pressure exerted on the head at birth.

**Etiology.**—The resistance of the mother's tissues, abnormal growth of hair upon the child's head, premature rupture of the membranes, and the use of forceps all predispose to this condition, which has, however, been observed in natural and uncomplicated labors. Joret observed cephalhematoma upon the occipital bone in children born in breech presentation. Pfeiffer reports 38 cases of cephalhematoma delivered in breech presentation and 15 in vertex presentation, and among them 4 forceps deliveries; 12 cases of breech presentation terminated spontaneously.

By *internal hematoma* is commonly meant an accumulation of blood beneath the internal pericranium. The existence of this condition cannot be demonstrated during life, but can be inferred from the existence of an external hematoma with pressure symptoms.

Hematoma of the sternocleidomastoid muscle occurs more frequently on the right side than on the left, and in breech than in occipital presentations. It is due to pressure and twisting of the head at birth. The hematoma is situated in the belly of the muscle and is very hard. It may be accompanied by rupture of some of the muscle fibres and inflammation of the muscle sheath.

FIG. 9



Rupture of diaphragm; intestine in thorax. Anterior view.

The prognosis is good, although several weeks may pass before the swelling has disappeared, and a slight torticollis may be present during that time. No treatment is required in this form.

Cases of *caput obstipum musculare congenitum*, or muscular torticollis, differ from simple hematoma of the sternomastoid in that they are caused by intrauterine malposition and pressure, which interfere with the circulation in the muscle and result in pressure atrophy.

Microscopically, the muscle shows interstitial myositis. Operation alone can improve or cure such cases.

**Treatment.**—In the treatment of cephalhematoma and hematoma of the sternocleidomastoid muscle it is of importance to notice whether the tumor is extending. In cephalhematoma we recognize the limitation of the tumor by the distinct edge showing the margin of the pericranium.

FIG. 10



Rupture of diaphragm; intestine in thorax. Posterior view.

As cephalhematoma is rarely double it will usually be found upon that one area of the cranium most exposed to pressure. Ordinarily, the tumor becomes sharply defined, does not extend, and does not increase in volume. Absorption usually goes on gradually, but very slowly, owing to the firmer condition of the tissues about the cranium. In cases where the tissue is edematous and where there has been injury to the soft parts with infection, a dressing kept moist with normal salt solution or boric acid solution should be applied. Should the tumor



increase rapidly in size or should absorption fail, the physician should incise the tumor freely, turn out its clot, examine thoroughly for the source of fresh bleeding, control such hemorrhage, and pack the cavity firmly with sterile gauze. This should be removed after twenty-four hours, the sac of the clot again irrigated, and a similar packing introduced. Aspiration of the clot has been advised, but this method is inefficient and unreliable.

### FRACTURES OF THE CRANIAL BONES.

The most important lesions present in cases where depressions of the fetal cranium exist are fractures. MacLennan<sup>1</sup> describes multiple fractures of the cranial bones as present in cases where superficial examination showed depression of the bones only. Bernheim<sup>2</sup> reports a case of delivery by forceps by which the meninges were injured and convulsions followed. Tissier<sup>3</sup> reports a case of breech presentation in which the forceps was applied to the after-coming head, followed by the gradual development of paralysis of the lower extremities. The nerves supplying the lower extremities must have been injured by forcible traction upon the limbs or by pressure over the lower portion of the trunk during delivery. Couvelaire<sup>4</sup> reports 51 autopsies upon children dying with head injuries immediately after birth; 18 of these children were at full term and 33 were premature. Among the premature infants there were 5 cases of cerebral hemorrhage. Among the 18 who went to full term there were 6 cases of spinal hemorrhage.

In reviewing these cases of direct injury to the cranium we find that the parietal bone is the one most frequently involved. Severe injury in this region may wound the branches of the middle meningeal artery, causing bleeding over the motor areas. The pressure of such a clot would lead to contracture and later to atrophy.

### INJURIES TO THE FACE AND SOFT PARTS.

Injuries to the face may also occur during labor and may result from severe pressure of soft parts against the bones of the pelvis or errors in operative procedure. Strassmann<sup>5</sup> describes a case of breech presentation where the finger was placed in the mouth to produce strong flexion when the head was born. The frenulum of the tongue was torn and free bleeding followed. In the same journal Wegscheider<sup>6</sup> describes a case where the introduction of the fingers into the mouth injured the gums in the posterior portion of the mouth, causing severe bleeding.

In some cases the tissues about the orbit may rupture during labor and suppuration follow. In a case under my observation the infant

<sup>1</sup> *Lancet*, 1902, vol. ii p. 632.

<sup>2</sup> *Ibid.*

<sup>3</sup> *Zeitschr. f. Geburtsh.*, 1903, Bd. xi p. 120.

<sup>4</sup> *Progres m&d.*, 1902, tome xiv. p. 179.

<sup>5</sup> *Ann. de gyn. et d'obst.*, 1903, tome lxx. p. 253.

<sup>6</sup> *Ibid.*

developed, several days after birth, a slightly opaque tumor at the site of the bruise, which, on incision, was found to contain a fluid full of leukocytes and microscopically resembling pus. The opening was prevented from closing for a few days, the cavity irrigated with sterile water, and it healed completely without injury to the eyeball or any scar.

**Injuries to the Eye.**—Jardine<sup>1</sup> reports a case of intracranial bleeding following labor in contracted pelvis in which the eye was injured and hemorrhage into the eye occurred. Keratitis followed and in twenty-four hours the cornea was opaque. It is sometimes difficult to decide whether bleeding from the conjunctiva has been caused by injury at birth or by some accident or manipulation. Wiener<sup>2</sup> reports a case of fatal hemorrhage from both conjunctivæ seven days after labor. In the absence of other causes he ascribes this to irritation produced by the employment of Credé's method to prevent ophthalmia. Terrien<sup>3</sup> describes wounds occurring in the eyes during labor by long-continued pressure, by the use of forceps or by unskilful manipulation with the hands. As a result of such injuries paralysis of the ocular muscles may occur, the lids may be paralyzed, and in deep wounds lesions may extend even to the eyeball. In some cases where the eyes themselves are not directly pressed upon, severe and continued pressure on the cranium may cause hemorrhage into the retina and choroid.

Paul<sup>4</sup> examined the eye-ground in 200 infants recently born. In those born after labor with contracted pelvis there was some hemorrhage in the retina in 50 per cent.; in children prematurely born spontaneously, in 40 per cent. In long and complicated labors of all kinds there was retinal hemorrhage in 40 per cent. In apparently normal cases of spontaneous birth there was retinal hemorrhage of greater or less degree in 20 per cent.

In injuries to the eyes, if the eyeball be dislocated, it should be replaced as soon as possible and kept bandaged with gauze pads saturated with boric acid solution or sterile water. An oculist should have an opportunity to advise regarding operative interference in all such injuries.

**Injuries to the Ear.**—The ear of the child may be torn from the head, or in cases where the base of the cranium is extensively fractured the temporal bone may be crushed and the auditory canal and tympanum lacerated.

In injuries to the ear if a portion of the ear be lacerated the tear should be immediately closed with fine sterile catgut. It must be remembered that in injuries to the ear a physician must always suspect damage to the mastoid region. Should infection occur, mastoid supuration will be threatened. In these cases the mastoid area should be opened early and drainage secured.

**Symptomatology.**—The immediate result of cranial traumatism at birth may be stupor or partial coma, the infant frequently lying quietly without crying, or in severe cases giving utterance to an irregular and

<sup>1</sup> *Journal of Obstetrics of the British Empire*, June, 1903, vol. iv.

<sup>2</sup> *St. Louis Medical Review*, April 25, 1903.

<sup>3</sup> *Inaug. Diss.*, Halle, 1900.

<sup>4</sup> *Arch. d'ophtal.*, 1903, tome iv.

sharp cry. In cases where severe internal hemorrhage occurs, symptoms of intracranial pressure will rapidly develop, with syncope and fatal issue. In cases of lesser injury the infant may rally immediately from the traumatism, and should it be able to nurse it may survive indefinitely. In these cases the secondary results of birth pressure will be observed later in contractures or atrophy of muscles with corresponding deformity. There will be alterations in sensation, in temperature, and in nutrition of the skin and neighboring tissues. (See Diseases of the Nervous System.)

The immediate symptoms accompanying injuries to the head in newborn infants differ somewhat from those produced by similar lesions in the adult. In the latter unconsciousness is the termination and may be the result of head injury. In the infant consciousness is scarcely developed at birth, and the physician cannot expect the same pronounced symptom which he would observe in the adult. In the adult it is unusual for the patient receiving a head injury to moan or cry. In the infant one of the most significant features of injury to the cranium or its contents is a peculiar, sharp, and almost incessant cry, very different from the crooning or grunting of the uninjured healthy infant. In the infant having a head injury at birth the breathing is seldom established normally. Usually, the respiratory rate is increased and the breathing is manifestly difficult. Where severe injury with intracranial hemorrhage is present the infant may be so overcome that the cry is feeble. Attacks of spasmodic breathing often accompany or follow the cry, with asphyxia in greater or less degree. The infant is often thirsty, taking water greedily or nursing with uncommon vigor. It is restless, with twitchings or convulsive movements of the limbs. The pupils may be widely dilated or contracted. The temperature may at first be subnormal, afterward rising considerably above the average; hence, the necessity for recording the temperature in all newborn infants. In some cases convulsions occur, usually developing some hours after birth and accompanied by asphyxia. Attacks of syncope with threatened failure of respiration and pulse are common, and in one of them the child may die. Death is often without warning and frequently without convulsive movements, the phenomenon of life ceasing almost instantly.

**Treatment.**—The treatment of injuries to the head occurring during labor should embrace especially a thorough prophylaxis, which is entirely obstetrical.

In all cases where such injuries are suspected the infant should be carefully examined as soon as possible after birth. The nostrils and mouth should be thoroughly cleansed to prevent asphyxia from the presence of mucus or other inspired material. All wounded surfaces should be thoroughly washed and kept protected by wet dressings. The scalp should be gently cleansed with sterile water and the cranium examined for evidences of depression. Should marked depression without evident fracture or distinct fracture and depression be observed, the physician must seriously consider the question of operation and a surgeon should be called in consultation. It must be remembered



that the cranial bones of the infant are very thin and yielding, that the tissues readily undergo necrosis, and, hence, that long incisions in the scalp or periosteum or meninges should if possible be avoided. If possible, depressed bone should be raised subcutaneously by introducing a blunt instrument through a small opening and cautiously raising the bone. If the fracture is in such a location that an important vessel is probably torn, then sufficient incision should be made over this point to give access to the vessel. The meningeal arteries are those most frequently injured and most often requiring ligation. It is well to drain such wounds for a short time to prevent the formation of a clot which may cause injurious pressure. While extensive operations cannot be borne by the newborn infant, it has been possible in a considerable number of cases to relieve pressure symptoms and to bring about the recovery of the infant by appropriate operation.

Ross<sup>1</sup> reports a successful operation twenty days after birth.

#### PARALYSIS FOLLOWING BIRTH.

**Facial Paralysis.**—The most frequent paralysis following labor is that of the facial nerve. This is seen most often in cases of difficult birth where pelvic contraction, excessive size of the child, or difficult instrumental delivery is present, and it has been seen in Cesarean section by Vogel,<sup>2</sup> who reports a case.

Facial paralysis in the infant may also be congenital and result in no degree from parturition itself. Franceschetti<sup>3</sup> reports 28 cases of congenital facial paralysis in newborn infants caused by some malformation of the bones of the cranium resulting in pressure upon the nerve at its point of exit or some abnormality of the nerve itself. Heller<sup>4</sup> describes similar cases. Köster<sup>5</sup> reports the case of 2 infants born of the same mother suffering from facial paralysis with total aplasia of the ganglion of the facial nerve. Mace<sup>6</sup> divides paralyzes following birth into traumatic and spontaneous. Other causes are amniotic adhesions compressing the nerve trunk or defective development in the facial nucleus. Libin,<sup>7</sup> in 3303 births at the Charité, saw 32 cases of facial paralysis, and of these 25 were delivered by the use of forceps and 7 occurred in spontaneous labor. The whole number of forceps deliveries was 1063. In 3 cases the facial paralysis was permanent. He ascribes this accident not so much to pressure by forceps as by the bones of the pelvis and contracted pelvis; meningeal bleeding; extravasation of blood in the region of the amniotic adhesions during the development of the face, and pressure of the child's cheek against the bones of the pelvis. Hemorrhages into the cerebral cortex, into the nucleus of the facial

<sup>1</sup> British Medical Journal, 1904, vol. i, p. 880.

<sup>2</sup> Zeitschr. f. Geburtshülfe und Gyn., 1902, Band xlviii, p. 474.

<sup>3</sup> Thèse de Bordeaux, 1903, 42.

<sup>4</sup> Deutsch. med. Woch., 1902, Band xxviii, p. 60.

<sup>5</sup> Inaug. Diss., Berlin, 1901.

<sup>6</sup> Thèse de Paris, 1908, 1361.

<sup>7</sup> Obstétrique, 1901, tome vi, p. 617.

nerve or nerve trunk, and pressure by bone and soft tissue are the usual causes.

In addition to facial paralysis the infant may develop strabismus or other ocular symptoms, as described by Nettleship.<sup>1</sup>

**Brachial Paralysis.**—Next to facial paralysis injuries to the brachial plexus may result in paresis or paralysis of the upper extremities. Thorburne found injury to the brachial plexus in 1 in 2000 cases; 50 per cent. of these were in breech presentation. The nerves most commonly affected were from the fourth to the sixth cervical nerves,

FIG. 11



Duchenne's paralysis of right arm before operation.

inclusive. Schüller<sup>2</sup> reports 3 cases of brachial palsy, 1 occurring in breech presentation. The roots of the sixth and seventh cervical nerves were implicated in some cases, and in 1 the sternocleidomastoid muscle was also shortened. Parry<sup>3</sup> describes 2 cases of paralysis of the arm and hand following delivery. In these the fifth, sixth, and seventh cervical nerves were at fault. One of these cases was complicated by torticollis and rupture of the fibres of the sternocleidomastoid muscle. There may be pressure on the nerves by a fracture and the resultant callus.

<sup>1</sup> Archiv f. Augenheilkunde, 1903, Band xlv, Heft 4.

<sup>2</sup> Wiener klin. Woch., 1902, Band xvi, p. 937.

<sup>3</sup> Lancet, 1902, vol. ii, p. 1631.

In a case reported by Rühle<sup>1</sup> the infant had paralysis of the right arm. Under treatment with galvanism recovery followed in five months after birth. In many of these cases separation of the epiphyses is suspected.

Stalper<sup>2</sup> draws attention to the fact that in many of these cases strong lateral traction is made upon the plexus, although an actual laceration of the nerves is of the greatest rarity. The tissues surrounding the nerves may be lacerated, and as a result a callus of connective tissue develops which compresses the nerve trunks. That extensive lesions

FIG. 12



Brachial plexus paralysis of right arm after operation; shows recovery of abduction of arm and flexion of forearm.

may follow injuries to the brachial plexus is illustrated in a case described by Philippe and Cestan;<sup>3</sup> in this patient bilateral monoplegia and muscular atrophy of the arms developed, with spastic symptoms and increase in tendon reflexes without diminution of sensibility. The lesions were those of the middle portion of the brachial plexus, and included the anterior as well as the posterior roots of the plexus. In long-continued spontaneous labor paralysis of one or both arms may result from cerebral

<sup>1</sup> Beiträge zu Geburtshilfe und Gyn., 1903, Band VIII, p. 64.

<sup>2</sup> Monatssch. f. Geburtshilfe und Gyn., 1901, Band XIV, p. 49.

<sup>3</sup> Gaz. des hôpitaux, 1900, tome LXXII, p. 785.

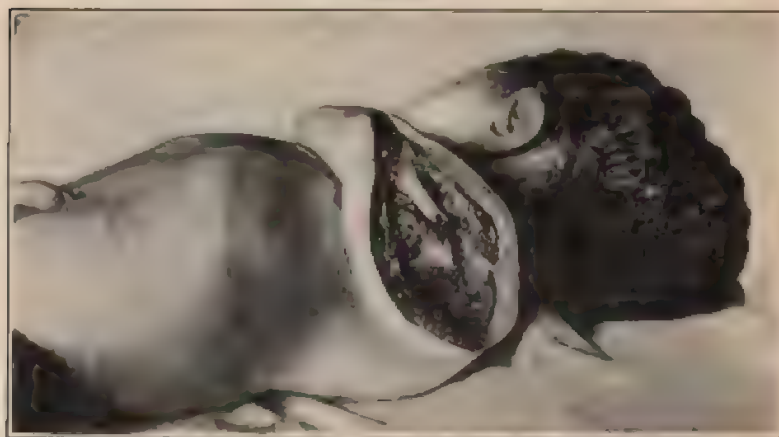
apoplexy, as has been mentioned. Martin<sup>1</sup> describes such a case, showing after birth inward rotation of both humeri, supination of the forearm, rigidity of the muscles, and increased reflexes. The injury present was an apoplexy in the motor centres which followed a short asphyxia.

**Treatment.**—Injuries to the brachial plexus have been usually treated by splints and by massage and the galvanic current. Care is necessary to keep up the warmth of the extremity supplied by the injured nerve. A splint may be needed if there is contraction. Where contractions have resulted Kennedy<sup>2</sup> has obtained good results by cutting down upon the injured nerve trunks, loosening adhesions, excising the injured and thickened portions of the trunk, and bringing the cut ends together with fine catgut. Of course, this cannot be done until some time after birth (Figs. 11 and 12). Very recently Clark, Taylor, and Prout<sup>3</sup> have done valuable work on these lines.

#### FRACTURES OF THE EXTREMITIES.

Fractures of the extremities are not infrequent in severe and complicated labors. Muus,<sup>4</sup> in 1200 cases of vertex presentation, found eighteen fractures of the clavicle, and in another series of 500 labors

FIG. 13



Fracture of humerus in difficult version.

four, making an average frequency of 1.3 per cent. The anterior clavicle was most frequently wounded in the proportion of 15 to 3. The site of fracture was usually the middle third.

Ragnvaldson<sup>5</sup> observed fracture of the humerus in spontaneous labor, vertex presentation, when the arm prolapsed beside the head (Fig. 13).

<sup>1</sup> *Lancet*, 1890, vol. 1, p. 541.

<sup>2</sup> *British Medical Journal*, 1903, vol. 1, p. 298, and 1904, No. 2286, p. 1065.

<sup>3</sup> *American Journal of the Medical Sciences*, October, 1905, p. 670.

<sup>4</sup> *Centralblatt f. Gynäk.*, 1908, Band xxvii, p. 689.

<sup>5</sup> *Ibid.*, p. 1208.

In some cases congenital fragility of the bones is an important element in the production of fractures. Officer<sup>1</sup> observed two such cases in the same family, one of whom had fracture of both femora and the other fracture of the arm at birth.

In a most important contribution to the subject of fractures in the newborn by Sperling,<sup>2</sup> he found by microscopic x-ray studies that many supposed fractures of fetal bones could not be referred to traumatism at birth, but that they result from abnormal development in the embryo. Amniotic adhesions are a frequent factor. These develop in the first or second month of embryonal life. They produce no callus, but an infiltration of small cells with periostitis. A skiagraph shows no bending of the bone in callus. Lesions in the skin at these points do not

FIG. 14



Congenital deformity of the hip-joint.

extend through the entire integument, but are superficial only and are also referred to amniotic lesions. Defects in the development of the fibula and in the digits were also found in these cases. In 60 per cent. of cases supposed to be intrauterine fractures these defects were present. There are no signs in these cases of previous traumatism.

Fractures of the femur are rare during childbirth. Injury to the ankle-joints may result from forcible traction upon the feet, from the slipping of the hand encased in rubber gloves.

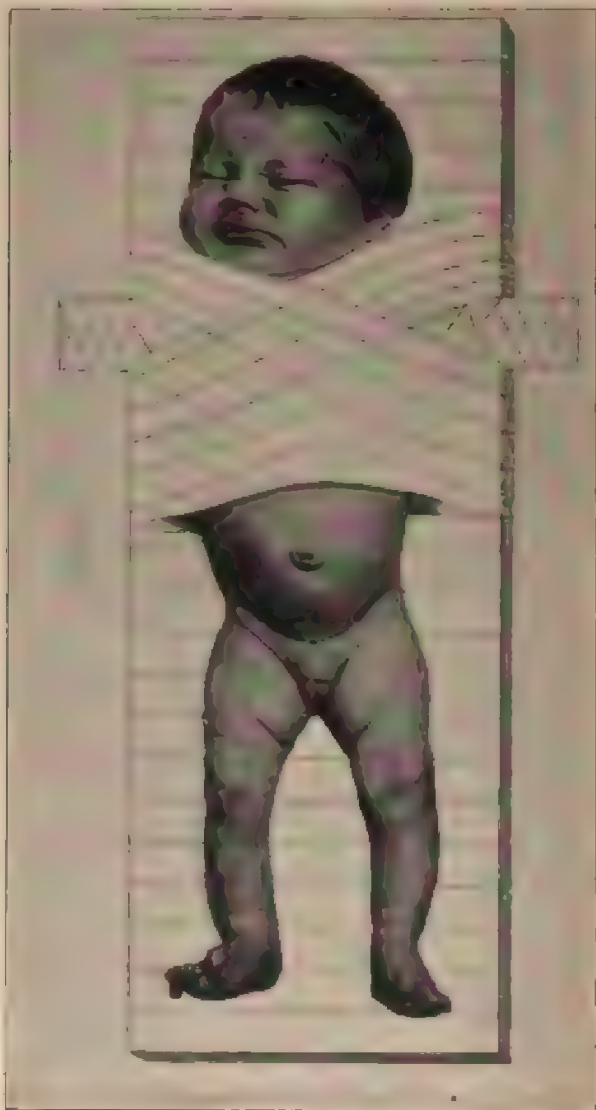
<sup>1</sup> Intercolonial Medical Journal of Australasia, October 20, 1902, vol. vii.

<sup>2</sup> Centralblatt f. Gynäk., 1902, Band xxvi, p. 1134.



Congenital dislocation of the hip-joints must not be mistaken for traumatic dislocation. The latter must be of excessive rarity, as an examination of recent literature fails to find it recorded. Whitman states

FIG. 13



Fracture of both clavicles: mode of dressing; recovery.

that congenital dislocation, "in some cases at least, is at birth a subluxation only, that becomes complete through muscular action."

Fracture of the ribs, sternum, or pelvis in the newborn may also occur.

PLATE I.



Fetal Skeleton, showing Failure of Ossification in Pelvic  
Centres.





PLATE II.



Fetal Skeleton, showing Failure of Ossification in Lower  
Extremity.



**Diagnosis.**—In diagnosing fractures of the extremities, ribs, sternum or pelvic bones the physician must remember that the epiphysis of a long bone may readily be separated from the shaft of the bone in the infant. Fractures are often green stick in variety and crepitus will be obtained indistinctly. The joints of the infant are so loose that they may be considerably stretched during delivery without actual trauma.

In an uncertain case of injury an x-ray photograph should be secured. (See Plates I. and II.)

**Treatment.**—The treatment of fracture of the clavicle in infants consists in keeping the infant as much as possible in a recumbent posture. It is difficult to apply a retention bandage or dressing to a newborn infant and equally difficult to keep the infant constantly recumbent. Some such device as that employed habitually by the Indian mother, who puts her infant upon a board and fastens it there with cloth or broad bandages, is appropriate in these cases.

In a case of double fracture of the clavicle occurring in the practice of Dr. Geo. A. Ulrich, of Philadelphia, the infant was put on a board twenty-four inches long and seven inches wide, carefully padded with a narrow board placed across it one-quarter of the distance from the top and firmly fastened. The infant's arms were carried upward, and were firmly bandaged to the sides, absorbent cotton being inserted so that the skin surfaces did not come in contact. A small, firm pad was placed between the shoulders and a similar pad over each clavicle. The splint was allowed to remain for eighteen days, when it was removed and the clavicles found to be completely united, with no perceptible deformity (Fig. 15).

In treating fractures of the long bones in the newborn care must be taken to avoid undue pressure in applying splints. Soft material which can be moulded to the limb of the infant should be selected. Ordinary pasteboard dipped in hot water may be softened and applied to the limb and then allowed to become partially stiff and moulded over the fracture. Spongipiline and other flexible material may be used. The splints should be carefully padded and the padding may be kept in place on the splint by covering it with gauze and stitching the gauze over the splint. The splint should be retained in position by bandages of gauze or flannel, which are more elastic than muslin bandages. So soon as a firm callus has formed massage is of especial value in these cases. By this adhesions are prevented, the muscles are developed, and absorption of the callus is promoted.

Compound fractures are of the greatest rarity and should be treated by drainage with wicks of sterile gauze and by thorough asepsis.

### INFECTIONS OF THE NEWBORN.

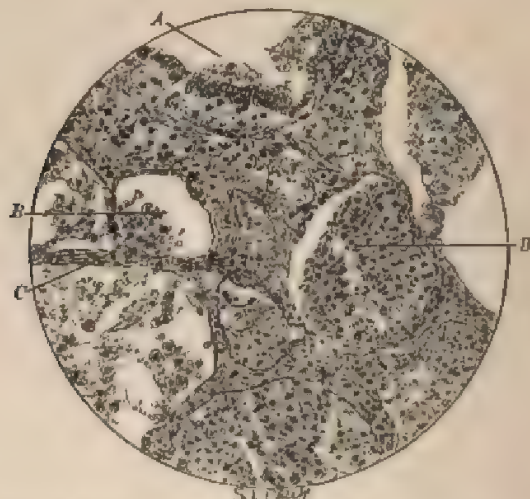
The infections of the newborn may be antenatal, those happening during birth and those arising immediately after birth (postnatal).

Syphilis is the most frequent of antenatal infections. Tuberculosis

is rare, only seven authentic cases being on record. Gonorrhea may occur as a prenatal infection in cases where the membranes were ruptured some time before delivery, or where a placentitis involved the amnion and gonococci thus found their way into the amniotic sac. In this way may be explained the cases of ophthalmia occurring in infants delivered by Cesarean section. Variola, scarlatina, measles, cholera, typhoid fever, yellow fever, relapsing fever, pneumonia, influenza, cerebrospinal meningitis, and malaria have all occurred in the fetus or newborn infant as a result of antenatal infection from the mother.

Maternal toxemia during pregnancy may be transmitted to the infant, with a fatal result within the uterus or soon after the infant is born. In one of my cases the mother became acutely toxemic before the birth of her infant, who perished eleven days after birth, with high temperature

FIG. 16



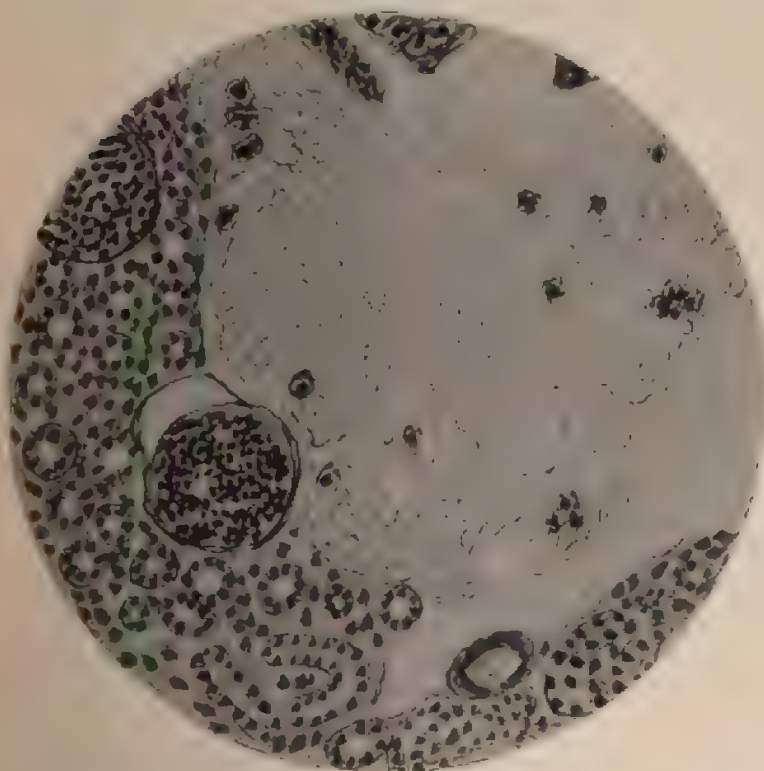
Lesion in the lung as observed in prenatal infection: A, wall of bronchus; B, air vesicles; C, connective tissue and intervesicular structure

and multiple hemorrhages from all mucous surfaces. At autopsy the umbilicus appeared free from infection or inflammation. The abdomen contained blood-stained serous fluid, the mesenteric bloodvessels were empty, and the lymph nodes were swollen. The pleural membranes were dry and sticky, the blood fluid and dark, the lungs dark red in color, the suprarenal capsules contained fluid blood, the kidneys were engorged, and multiple hemorrhages were present in the stomach and intestine. At the time of the infant's death its hemoglobin was 110 per cent., hemoglobin crystallizing upon the slide. The red corpuscles were greatly distorted and the eosinophile cells were much increased. The infant's feces contained bacillus coli communis and staphylococcus pyogenes aureus. The different organs showed non-infective periarteritis in all the small vessels. I have seen similar lesions in an infant born just after

the death of the mother from eclampsia, the infant surviving the mother for some days and dying with symptoms of acute toxemia (Figs. 16, 17, and 18).

**Treatment.**—Practically the physician must recognize conditions accompanied by passive hemorrhage without anatomical lesion in newborn infants as cases of infection. As pathogenic bacteria are found in the intestine of the infant in many of these cases, the most valuable method of treatment in my experience has been the thorough irrigation of the intestine with sterile salt solution. The infant's food must be carefully chosen and suitable stimulation given. No other method of treatment has been of especial value. The treatment of syphilitic and gonococcal infections will be mentioned later.

FIG. 17



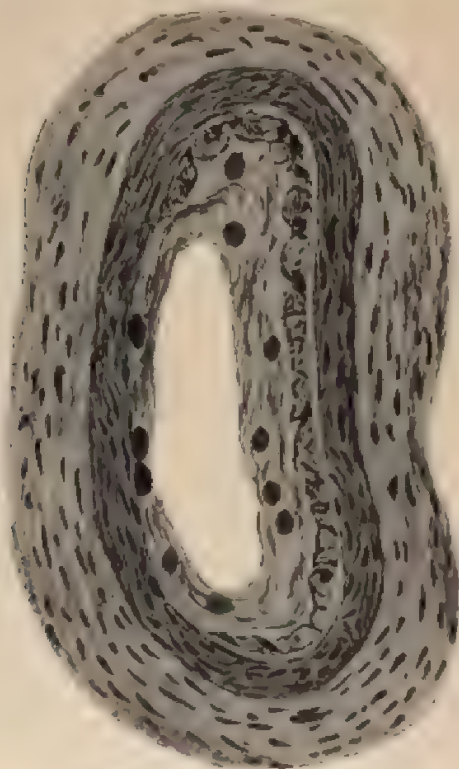
Lesions in kidney; prenatal infection.

**Intranatal and postnatal infections** may occur by means of the vaginal secretion of the mother, the hands of the physician or nurse, dressings, instruments, clothes, the air of the room, water used for bathing or drinking purposes, and the infant's food, whether human milk or cows' milk. Finally, cases of autoinfection by means of the infant's own secretions (nasal, buccal, vaginal) have been reported.

The bacteria which has been isolated from cases of general infection of the newborn are: the streptococcus, staphylococcus albus, citreus, and aureus; pneumococcus, an organism closely resembling it, pneumobacillus of Friedländer, bacillus coli communis, Gartner's bacillus, bacillus proteus vulgaris, and bacillus pyocyaneus. The gonococcus, diphtheria bacillus, and tetanus bacillus have been found locally at the point of infection.

The most common point of entrance is the umbilicus, although the skin, the mucous membrane of the respiratory, digestive, and genito-

FIG. 18



Lesion in artery; prenatal infection.

urinary tracts, the conjunctiva and the ear may all act as starting points for local or general infections. The mammary glands may be infected as the result of squeezing or rubbing.

Staphylococci (albus and aureus) have been found in woman's milk from an apparently normal breast; in cases of irritation or fissure of the nipples these organisms are almost invariably present, and streptococci have been found as well.

**Symptomatology.**—The symptoms of septic infection in the newborn naturally manifest themselves in the organ where infection originates.



Infection of the skin may result in erythema, pemphigoid eruptions, furuncles, abscesses, ulceration, and petechial hemorrhages. If the mouth be the site of infection, there may be catarrhal stomatitis, thrush, superficial or deep ulceration, pseudomembranous inflammation, and even gangrene. Gonorrheal ulcers are occasionally seen in the mouth. Smaller or larger ecchymoses on the palate and inside of the cheek are common. The cervical lymph nodes may be swollen.

Infection of the respiratory tract results in nasal catarrh, laryngitis, bronchitis, or pneumonia.

Anorexia, vomiting, diarrhea, and hemorrhage from the stomach or intestines follow infection of the gastrointestinal tract. While such infection may undoubtedly be primary and prove the starting point of a general sepsis, the majority of cases of gastrointestinal infection are secondary to the entrance of bacteria at some other point.

Infection of the vagina may be followed by mild or severe inflammation, and by gangrene.

The urine may be diminished, and hemoglobin, blood cells, pus, and casts may be found in it.

Pus may develop in one or more joints. There may be progressive loss of weight, and the influence of toxins on the nervous system may cause restlessness or coma, local or general convulsions, irregular pulse and irregular respiration. The temperature throughout the entire course may not be elevated, or it may be very high.

*Ophthalmia*.—If the eyes have been infected the lesions will differ with the variety of the infecting germ and its virulence. Zabel<sup>1</sup> examined 33 cases of acute typical ophthalmia, finding the gonococcus present in 19. The pneumococcus, staphylococci, and bacilli were found, and in some cases no pathogenic bacteria were present. Lesions of the cornea do not prove gonorrheal infection, for in 6 cases without the gonococcus the cornea was injured. In gonorrheal ophthalmia the conjunctiva is slightly reddened at first and a profuse secretion of thin, glairy mucus is formed. This soon changes to pus, often of a bright-yellow color, and the tissues become a deeper and brighter red. Should the infection proceed unchecked the pupil will be contracted, the cornea will gradually become cloudy, iritis and adhesions will develop, ulcer and perforation of the cornea may result, pus may form in the anterior chamber, and the eye may be lost. Catarrhal conjunctivitis may follow the subsidence of acute symptoms.

*Tetanus Neonatorum*.—Symptoms caused by toxemia of the tetanus bacillus commonly appear during the first and second weeks, rarely later than the third week. Trismus is the predominate symptom, followed by spasms of the muscles of trunk and extremities, dysphagia, dyspnea, cyanosis, cardiac failure, and incontinence. Death is the usual ending.

*Duration*.—Ordinarily the infections last from two to five days, though cases of less than twenty-four hours' duration have been reported, and others may linger two weeks or more.

*Prognosis* is very grave. Cases which do not terminate in rapid death may go on to athresia, chronic digestive disturbances, and severe anemia.

<sup>1</sup> Inaug. Diss., Halle, 1903.

<sup>2</sup> Lancet, 1903, vol. II, p. 103.

**Pathological Histology.**—There is always parenchymatous degeneration of the organs, and often the liver shows fatty changes as well. Hemorrhages into the skin and mucous membranes, as well as underneath the pleura, pericardium, and Glisson's capsule, are very common. Swelling of the spleen and lymph nodes, pneumonia or pulmonary congestion are the rule. Thrush in the mouth and esophagus and ulcers at any point in the intestinal tract are among the less frequent lesions, while pus in the umbilical vessels and in the liver, as well as on any of the serous membranes, may be found.

**Treatment.**—The treatment of the various infections of the newborn varies with the organ infected. At the umbilicus, in the mouth and the nostrils local antiseptics may be attempted with a fair prospect of success. It must be remembered that infants are very susceptible to mercurial and carbolic poisoning, and hence very dilute solutions or mild antiseptics only should be employed.

Boric acid or salt solution may be used freely and usually without injury. Care must be taken not to spread infection by injuring the tissues through harsh manipulation. Gentle irrigation, as the use of a spray, is especially valuable.

The breasts of the newborn may be dressed with sterile gauze compresses soaked in boric acid solution. They must not be handled nor squeezed.

In pulmonary infection antiseptics is impossible and the infant must be treated by supportive measures only. Oxygen may be inhaled and it is necessary to give artificially digested nourishing food at intervals and as much alcohol as the infant can possibly digest. Restlessness and fever are best controlled by the external application of cold by either sponging or the use of compresses.

In infection of the intestine I believe in the importance of free irrigation with salt solution. The free use of water as a drink is important in all intestinal infections.

In diphtheria the value of serum by injection is established. In tetanus the value of serum is uncertain as it is used after toxemia has developed. Chloral hydrate in doses of 0.06 gm. (gr. j) by mouth, or rectum, repeated every few hours is of positive benefit and may be curative in late cases. Warm baths have a sedative effect.

The value of Credé's prophylactic treatment in ophthalmia has of recent years been much discussed.

Kraenzkamp,<sup>1</sup> among 4500 children with whom Credé's prophylactic treatment was carried out, saw ophthalmia in but 11 cases, of which 2 only were severe. Some prefer the use of argyrol in 10 per cent. solution as being absolutely without danger. Rosner<sup>2</sup> obtained the best results by using within the eye 10 per cent. solution of protargol and cleaning the lids with a 3 per cent. solution of boric acid.

Bischoff<sup>3</sup> observed symptoms of irritation in 80 per cent. of cases in

<sup>1</sup> Inaug. Diss., Halle, 1903.

<sup>2</sup> Wiener med. Blätter, 1903, Band xxvi., No. 16.

<sup>3</sup> Centralblatt f. Gynäk., 1903, Band xxvii. p. 293.



which silver was used. This, however, subsided in four days without injury. Protargol seemed as irritating as silver nitrate in his experience. Acetate of silver seemed less objectionable. He considered the silver irritation of no practical importance. Veverka, among 1100 children treated with protargol, observed but 4 cases of ophthalmia, and these were secondary infections. The possibility of infection attacking the tear-duct of the newborn has been reported by Heimann.<sup>1</sup> Additional testimony to the value of the acetate of silver is given by Scipiadès.<sup>2</sup> He treated 200 newborn children with 1 per cent. solution of acetate of silver without the development of ophthalmia. In 11 cases the remedy caused free secretion.

The majority opinion is distinctly in favor of the use of some preparation of silver in the eyes of newborn children in maternities. Whether this be Credé's method as originally proposed, or the use of argyrol or protargol in from 1 to 10 per cent. solution, or acetate of silver, or saturated solution of boric acid, or equal parts of salt solution and boiled water, must be left to the judgment of the responsible physician. Personally, I have seen good results in hospital practice by giving all mothers a preliminary vaginal douche of lysol and green soap, and by using boric acid solution in the eyes of the infant. In private houses, with patients of known character, I have not always found it necessary to employ Credé's prophylactic treatment.

When ophthalmia develops the most prompt and vigorous treatment is necessary. The child should be isolated in charge of special nurses. In the acute stage cold should be applied constantly but very carefully with small compresses taken from a cake of ice.

Silver preparations may be dropped into the eye, followed by salt solution. If argyrol or protargol be used, salt solution is considered unnecessary. Irrigation is of decided value. A fountain syringe in whose tube is placed a small glass pipette like a medicine dropper, delivers a small stream of antiseptic fluid without force. The infant is placed across the nurse's lap with the infected eye lower than the other. The non-infected eye should be protected by sterile gauze or cotton and bandage. The fluid is allowed to run from the inner canthus downward and outward, thus avoiding infection of the healthy eye. I have found alternate irrigation with 1:8000 bichloride solution and saturated solution of boric acid, using one of these every two hours and each of them every four hours, to be of great benefit. The eyes should be very gently dried with sterile linen or gauze after irrigation. Should the pupil be contracted it should be dilated with atropine. If the case improves, treatment should be made less frequent, the silver may be discontinued and bichloride omitted, and irrigation with boric acid solution or salt solution will be sufficient. If the infant seems depressed by the use of cold and the redness subsides, the use of ice may be omitted. It should in any case be discontinued as soon as possible.

It is difficult to give the precise statistics of the results of ophthalmia.

<sup>1</sup> Deut. med. Woch., 1903, Band xxix, p. 86.

<sup>2</sup> Samml. klin. Vortr., Leipzig, 1902, No. 345.

In my experience considerable and permanent damage to the eye is rare. The complete loss of the eye is seldom observed.

The physician should not forget to warn nurses and attendants of the danger of contagion.

**Icterus.**—Idiopathic or primary icterus in the newborn infant is due to some disturbance of the physiological rearrangement of functions which takes place immediately after birth. Whether the fault lies in the blood or in the liver has not been proved as yet. Jaundice is recorded in newly born infants in 35 to 75 per cent. of the cases.

Secondary icterus may be one of the symptoms of a general sepsis, or it may be due to interstitial hepatitis (usually of syphilitic origin), to obliteration of the bile-ducts, or to blocking of the common duct by catarrhal inflammation or rarely by a calculus.

In the congenital cases Griffith<sup>1</sup> inclines to the view that there is a failure of development. The course of the disease is not acute.

**Prognosis and Treatment.**—The primary cases recover spontaneously. Of the secondary cases, those due to sepsis are often fatal, those due to obliterated bile-ducts invariably so.

Where there is a catarrhal obstruction simple saline aperients are sufficient to relieve the symptoms. Jaundice associated with syphilis clears up with mercurial treatment. In cases of jaundice due to congenital occlusion of the bile-ducts treatment is entirely symptomatic and almost entirely without result.

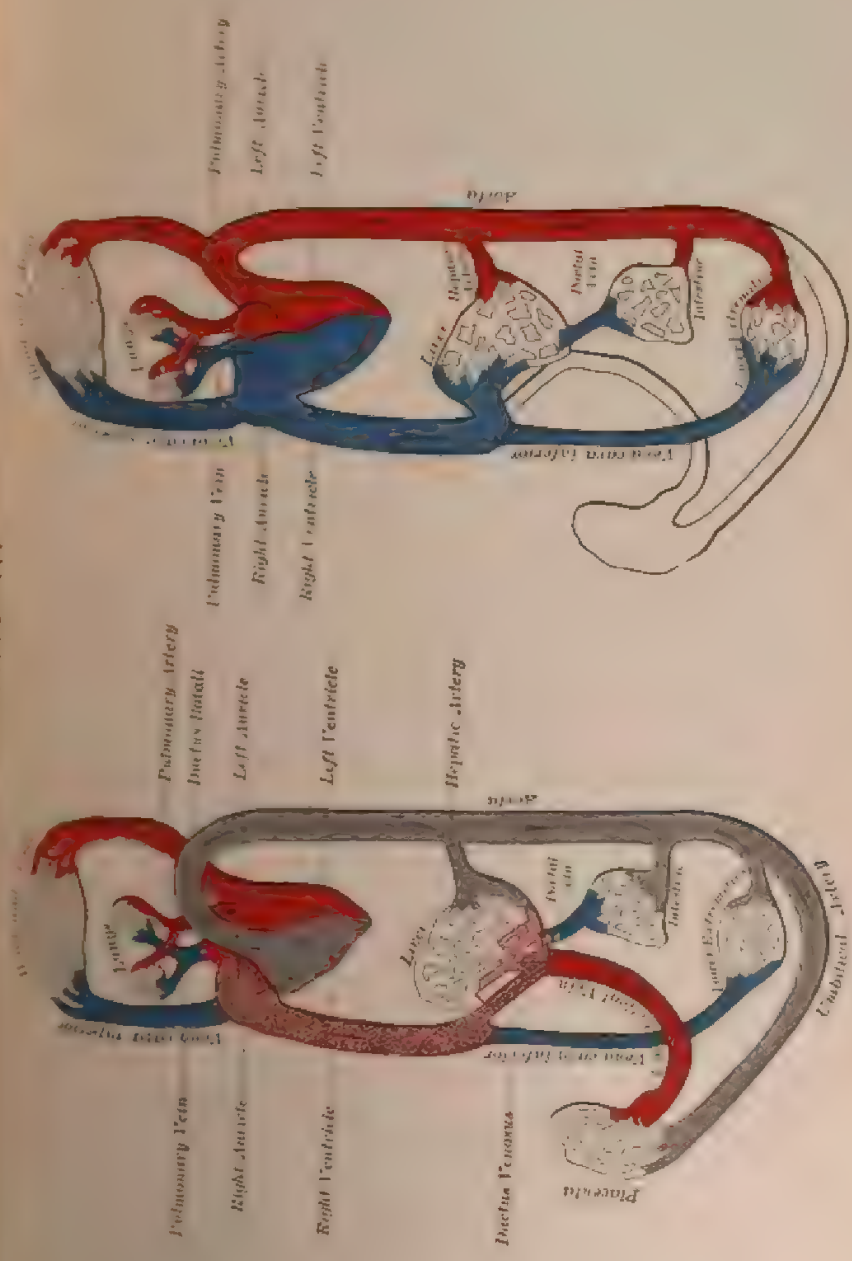
**Pemphigus.**—Pemphigus is an infection of the skin not infrequent in the newborn. It may be distinguished from syphilitic pemphigus from the fact that it does not attack the soles of the feet and the palms of the hands, that it is contagious, and that it yields to local treatment. *Staphylococcus pyogenes albus* and *aureus* are the organisms almost invariably found in the fluid of the bullæ which characterize the disease. General symptoms may or may not accompany or precede the eruption. Mild antisepsis of boric acid, sterile dressings, and careful feeding and stimulation are effectual.

**Syphilis.**—The infant may be apparently healthy at birth, and characteristic symptoms may not appear until several weeks later. On the other hand, severe cases may show an eruption at birth, consisting of papules, pustules, or bullæ, especially numerous on the palms and soles. The contents of the bullæ are often blood-stained. Emaciation and a general appearance of old age accompany the eruption. Soft cords with or without hemorrhage are common in these cases.

In infants who present no symptoms at birth the characteristic snuffles, fissures of the lips and anus, excoriation of the buttocks, and eruption first apparent on the face and hands may not develop for two to six weeks, but they are usually present within two months after birth. Hemorrhages from the mucous membranes are very common.

**Treatment.**—This is the usual treatment detailed under the special heading on Syphilis.

<sup>1</sup> Archives of Pediatrics, April, 1905, p. 257.



A. Diagram of Fetal Circulation Before Birth. (Modified from Hasse.)  
 B. Diagram of Fetal Circulation After Birth.



## SECTION II.

# DEVELOPMENT, GROWTH AND HYGIENE.

By LEROY MILTON YALE, M.D.

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### CHAPTER IV.

#### CHANGES AFTER BIRTH—HYGIENE OF THE INFANT AND NURSERY.

To further our knowledge of the conditions intimately associated with growth and development, it is essential that we should revert briefly to some of the organs of the infant that were studied in the previous chapters and which we must now regard from their physiological standpoint.

At the beginning of extrauterine life changes take place in the circulation which are best understood by an inspection of the plate (Plate III.) of the circulation of the blood through the vessels of the placental attachment before the infant begins its separate existence.

**Circulation.**—The circulation of the fetus up to the institution of respiration and the cessation of flow through the umbilical cord is in brief as follows: Red blood enters the fetus through the umbilical vein. Beneath the liver the vein divides; one portion, carrying the larger part of the blood, enters the transverse fissure. This blood, already somewhat mixed with darker blood, reaches the vena cava by way of the hepatic veins. The smaller current goes directly to the vena cava through a continuation of the umbilical vein, called the ductus venosus. This blood, still largely red, meets in the cava blood returning from the abdominal veins. This mixed blood is still, however, as nearly arterial in character as any sent to any organ, except the liver, during fetal life. In the right auricle it meets the blood coming from the superior vena cava. In earlier fetal life it is thought, from the anatomical structure of the heart at that time, that the two currents do not mix very much, that from below being directed by the Eustachian valve through to the foramen ovale to the left auricle. The flow coming back from the undeveloped lungs very slightly alters the character of the blood current. It is then thrown by the left ventricle through the aortic arch, the carotids, and subclavians to the head and upper extremities. These

parts are, and probably consequently, far more developed than the rest of the body, more especially in the early and middle period of fetal life. The current from the superior vena cava, however, probably descends directly to the right ventricle, and is thrown into the pulmonary artery. The branches of this artery cannot distribute much blood to the still unexpanded lungs, and the bulk of the flow is diverted to the ductus arteriosus (which is, in effect, a branch of the pulmonary artery), and delivered into the descending aorta just below the arch. As the fetus nears term, however, the anatomical changes hinder the free flow through the foramen ovale, and the course of the blood approaches more nearly the postnatal. The lower extremities, therefore, for the most part, receive blood which has already done duty in the upper extremities and in the head. This is probably the main reason of their relatively small size. The return circulation to the placenta is by way of the internal iliac, hypogastric, and umbilical arteries. The distinction between arterial and venous blood, so readily recognized after birth, does not exist in fetal life after the placental blood has entered the liver, but the two sorts are mixed in various degrees in different parts of the body. (See Plate III.)

**Changes at Birth.**—As soon as the respiration is thoroughly established the circulation of blood is greatly increased in the lungs. With the functioning of these organs, the blood returning from them is no longer dark. As soon, too, as the respiration is established the flow through the umbilical vessels is usually arrested by ligation, and umbilical veins and the ductus venosus fill with clots, and usually within a few days are practically obliterated and become fibrous cords. That part of the umbilical artery which within the abdomen of the infant is called the hypogastric artery is also obliterated, save a small branch to the bladder.

The changes in connection with the circulation within the heart itself are more important clinically, because of cardiac symptoms associated with the persistence of fetal conditions. The ductus arteriosus disappears by the same obliterative process as the umbilical vessels and ductus venosus, and is closed usually within ten days after birth. The foramen ovale, which, as already mentioned, does not carry a large amount of blood in the latter part of fetal life, becomes still more obstructed after birth by the growth of a valve-like flap. Although the closure remains not absolutely complete in very many cases for months after birth, it is nevertheless functionally adequate very shortly after birth. Persistent patency of the foramen to a degree to cause symptoms is, nevertheless, not a very rare condition.

The activity of the circulation diminishes with the growth of the body, and probably necessarily; for while the weight of the heart bears about the same proportion to the body weight in the early years as in adult life, the capacity of the vascular system and the length of the vessels to be served diminishes the rapidity of the circulation. Similarly, with the increase in body length, the pulse rate falls. This rate is very variable in individuals; females have a higher rate than males, and



in the same individual it may vary rapidly from time to time, especially in very young children. Observers give very different estimates for different ages. Holt's table gives the lowest figures, but he states that they were taken when the infant was asleep or perfectly quiet, which the others do not. Holt gives:

Six to twelve months . . . . .	105 to 115 per minute.
Two to six years . . . . .	90 to 105 " "
Seven to ten years . . . . .	80 to 90 " "
Eleven to fourteen years . . . . .	75 to 85 " "

It is quite certain that the pulse rate is less if counted by a nurse to whom the child is accustomed than if taken by the physician, who is not so familiar a person. The physician who is a stranger must expect higher rates than when he is acquainted with the child.

**The Stomach.**—The capacity of the stomach is quite variously estimated by different writers, and probably because of the different methods employed. It is not perfectly easy to determine when the stomach is normally distended and not overdistended. Holt's summary of his inquiries is: "In brief, the average capacity was, at birth, one and one-fifth ounces; at three months, four and a half ounces; at six months, six ounces; at twelve months, nine ounces." Rotch's estimates are, however, considerably smaller. Whatever estimates are accepted, they will give no justification for the enormous quantity of liquid food often fed to infants at one meal. The stomach is more directly a part of the intestinal tube than in the adult.

**The Intestines.**—Probably the most important fact regarding the intestines to the practitioner is the great relative length and very pronounced S-shaped curve of the sigmoid flexure. This peculiarity has to be borne in mind when the colon has to be irrigated, and instances have been reported in which the tardy emptying of this prolonged flexure has led to needless interference for supposed imperforation. The intestines are liable to distention to gas in babies who are artificially fed, and the pressure of the gas may still further change their position.

**The Liver.**—The liver is notably large in infancy, being at birth proportionally about two-thirds larger and for the first three years of life still at least one-third larger than in the adult.

**The Bladder.**—It is necessary to note that the bladder in infancy may occupy a much higher position than in adult life. Being quite distensible after the earliest weeks, and having quite lax pelvic attachments, it easily rises above the pubis and may be a source of confusion to the diagnostician, as well as to the operator.

**The Special Senses.**—It is doubtful whether any exist at birth. The only one which I have been able to see evidence of is *smell*, and this chiefly from the behavior of the infant when, if really awake, it is placed upon the breast. The infant's eyes certainly are affected by strong light at birth, as evidenced by the closing of the lids against it. Later, it seems to be interested in gazing at illuminated objects. But *sight* in the ordinary sense of distinct vision cannot occur, except occasionally



by accidental adjustment, until the co-ordination of the ocular muscles is established, which may require several months, possibly five or six. *Hearing* is developed fairly early, usually within six or eight weeks. In my opinion much that has been written about early hearing is due to its confusion with the recognition of concussion, the infant being startled by the jar, not by the sound, and being disturbed nearly, if not quite, as easily by the former when noiseless. It is, of course, not intended to deny the great sensibility of young children and even infants to sound, both as to degree and quality, after the hearing is well established. It is claimed by some that taste is developed very early, if indeed it be not present at birth. I have been unable to verify the latter claim. But there is no doubt that quite early differences of taste are recognized. It is not easy to decide how early, because some things not agreeable to adults or older children do not give offence to little children. Pungent substances are usually objectionable to children, and I have had doubts to what degree the irritation caused by such articles gave rise to an appearance to a dislike for the taste of them. General bodily or *tactile sensibility* is not very acute in the newborn; nor is sensibility to pain; but both sorts of sensibility are rapidly developed.

**The Muscular System.**—The development of the muscular system is more striking as regards gain in co-ordination than in actual gain in power; for example, the chance blow delivered by the infant's hand or foot demonstrates considerable muscular power long before the child can co-ordinate its motions. Automatic closing of the hand upon an object occurs very early, but not until after three months will it be likely to purposely grasp anything. It will be at least another three months before it can sit upright. Soon after this feat is accomplished it may learn to creep or to hitch itself along in a sitting posture upon the floor. With the last quarter of its first year usually come attempts at climbing up beside chairs or the parent's knee, the time of standing gradually increasing until it can stand alone. Great variation within strict limits of health exists as to all these developments, and particularly as to walking without aid and habitually. This usually occurs within the first quarter of the second year. Debility from any cause may retard it. Overfatness from peculiarities of feeding may increase the difficulty of balancing, and so hinder the free walk of a child who has been for some time able to stand with support. No urging of the baby should be permitted. Granted ordinary mental development, it will walk as soon as it properly can, but it must have wide-toed slippers or shoes that will not cramp the feet and add to the difficulty in balancing. Defective children are, of course, not here considered.

**Speech.**—Speech is developed very differently in different children. Not only does the first attempt to speak vary considerably in time, but the method of development differs. Given apparently equal mental development and equal ability to understand speech, one child will articulate with great clearness, while another will pour out a great flow of conversation, the meaning of which only the initiated or the very

imaginative admirers can guess. Occasionally, a child is met with who is evidently intelligent, who seems to shrink from speech until it can speak well. But, as a rule, intelligent children make some attempt by the completion of a year and "put words together" during the second. Occasionally, one speaks with precision at two years of age, and I recall one of this age who rolled out with phonographic accuracy the sonorous Greek lines of the "Iliad." Owing to the almost automatic position of the tongue-tip in making their sounds, *p*, *m*, *t*, and *d* are usually the first articulated consonants, and as a consequence "papa," "mamma," "dad" are among the first words and have become accepted as endearing names in most tongues.

### GROWTH OF SPECIAL PARTS.

In addition to the foregoing a few words may be said in regard to the proportions and peculiarities of some parts and regions of the body, both because upon these local changes the general changes depend and because the local changes are intimately connected with important physiological facts.

**The Head.**—The head participates in the general rapidity of growth of the first year, so that at the end of twelve months its circumference is almost a third larger than at birth, and quite a third larger at a year and a half. The increase in circumference is thereafter slower, say 5 cm. (two inches) from eighteen months to five years, and 2.5 cm. (one inch) or thereabouts during the succeeding ten years. But the size of the head at puberty and thereafter varies very much in proportion to the body height. At puberty the average circumference is rather more than one-third of the total height, at the completion of growth rather less than one-third.

With this growth marked changes in proportion occur. At birth the face is noticeably small as compared with the cranial vault, so that the centre of the vertical diameter is at the top of the orbit instead of at the pupil, as in the adult. The mandible does not descend into the prominent chin of maturity, and is but little below the occipital condyles. So that infancy bears a remarkable resemblance to toothless age. The ridges of the skull are little marked, the frontal and parietal prominences decidedly so. The bones of the vault have no diploë. Of more clinical importance is the fact that the sutures exist only potentially in the approximating margins of the component bones. The frontal or metopic suture still exists. The parietal bones are incomplete, their missing anterior corners and the divided frontal leaving the space known as the anterior fontanel, usually about 3.81 cm. (one and one-half inches) anteroposteriorly and one inch transversely. A similar defect at the approximation of the posterior angles of the parietals to the occipital makes the posterior (triangular) fontanel not above one-half the size of the anterior. The other fontanels are scarcely of clinical importance. Nor is the composite nature, at this age, of the occipital

and temporal bones. But the absence of the mastoid process is worth remembering as bearing upon aural diseases. The osteal development of the cranial base and the gradual fusion of the centres of ossification are very interesting, but of moderate clinical importance. It may be worth while to remember that the fusion of the basal parts of the occipital and sphenoid is one of the last to take place, only in fact when the organism is practically mature. The closing of the anterior fontanel, which occurs on the average at about the age of eighteen or twenty months, is clinically the most important of all these cranial bony changes. The changes and distortions of the skull as the result of disease or dyscrasia do not belong to this section.

The amount of hair upon the scalp at birth may vary from complete baldness to exuberance, but it is usually scanty. This hair is generally lost within a few weeks and is replaced by another growth, ordinarily of a different color from the first.

**The Chest.**—The chest in the newborn is noticeably small. So long as the placenta performs the respiratory function of the lungs, the latter remain only as reserve or accessory organs, the lung tissues growing to meet their prospective use, but the air vesicles not expanding. Hence, the small size of the chest, which is usually at birth rather less in circumference than the head and still less when compared with the abdomen. By the age of two years the circumference of the chest usually is as great as that of the head, and thereafter gradually and increasingly gains proportionally. If it be remembered that the average gain in head circumference is not much more than an inch between five and fifteen years, while that in chest circumference is about ten inches, this relative change of proportion will be emphasized.

**The Abdomen.**—The abdomen at birth is ordinarily larger than the chest and even a little larger than the head, but the rapid growth of head and chest very soon destroys this predominance and the whole trunk varies little in its circumferences during infancy, so that "baby has no waist" is a nursery axiom. In the periods of second infancy and adolescence the circumference of the chest gains upon that of the abdomen, the relative size of the latter varying much according to diet and tendency to fat.

**The Spinal Column.**—The fact regarding the spinal column of an infant which first attracts attention is its great flexibility, especially in the anteroposterior direction. This persists to a very great degree, however, through childhood and even to adult life. So prominent is this that any spinal rigidity in a child should at once be investigated as evidence of disease, probably of the column itself. In the newborn this flexibility of the spine is so great that the distinctive curves do not exist, save that the occiput and sacrum present permanent posterior convexity. Between these the spinal column usually falls into a single long curve, the direction of its convexity being determined by the position in which the child is placed or held. As the lower extremities become more and more placed in the position of extension, the traction of the iliopsoas muscles helps to give a lumbar curve with forward con-

vexity. As the child learns to stand and walk this curve calls for a compensating dorsal curve in balancing, the development of the chest assisting in its formation. The total curve begins thereafter to resemble that seen in the developing child, save that the cervical curve is less evident. The infant is often said to have no neck. In fact, the proportion of the cervical vertebræ to the whole column is not so very much different from what it is later, but the region is enveloped in fat or in loose integument and hidden behind and masked by the relatively enormous head, so that the occiput seems to rest upon the shoulders.

**The Teeth.**—At birth the teeth, both of the temporary and permanent sets, exist rudimentally in the jaw. As has already been noted, the mandible has little angle at birth, yet its gum does not quite touch that of the upper jaw. The temporary or so-called milk teeth begin their development early in fetal life, the process advancing slowly and continuing long after their eruption through the gums. Very rarely, indeed, teeth are found to have pierced the gum at birth. In a case in my own practice such a tooth proved to be a supernumerary one. The infant's mother and maternal grandmother each had a supernumerary infantile incisor persisting among the permanent teeth. The infant's tooth, however, proved to be deciduous and was replaced, contrary to the rule, by a supernumerary incisor in the permanent set.

While the average time of the eruption of the teeth is pretty well agreed upon, individual variations are very wide within the limits of apparent good health. In artificially fed children in dispensary practice the average date, tooth for tooth, is later than in breast-fed infants. Whether this difference exists under well-managed artificial feeding I have not enough statistics to state, but I am inclined to think that a slight difference still exists as compared with the best breast nutrition. I also place a good deal of stress upon family peculiarities—paternal quite as often as maternal. In some families even with good breast feeding and evident good nutrition the teething will be late. On the other hand, I recall in a forward teething family a child cutting teeth in advance of the average, in spite of very poor nutrition and pronounced infantile scurvy. This child's father I had also had under observation as an infant. He cut his first tooth at four months and had sixteen teeth at sixteen months. It should always be kept in mind in tracing hereditary peculiarities that the child is not to be compared with its parents, but its parents' childhood.

The temporary set contain twenty teeth—ten upper, ten lower—namely, in either jaw four incisors, two canines, four molars. While the order of their eruption varies, so that this is differently stated by different writers, the majority agree that the time and order of eruption are nearly as follows:

#### ERUPTION OF TEMPORARY TEETH.

Lower central incisors . . . . .	6 to 9 months.
Four upper incisors . . . . .	8 to 12 "
Lower lateral incisors and four first molars . . . . .	12 to 15 "
Four canines . . . . .	18 months to 2 years.
Four second molars . . . . .	2 to 2¼ years.

It is not rare for dentition to begin considerably earlier than the earliest date just given; but for some reason, probably the disturbance of weaning, the date of the coming of the teeth in the third and subsequent groups are, in my experience, less likely to be anticipated. In each group, except the incisors, the lower teeth usually precede the upper (Fig. 19).

All the teeth of the temporary set have successors in the permanent set, the germ of the latter being attached behind the sac of the former. To these are added three pairs in either jaw. The first of the permanent

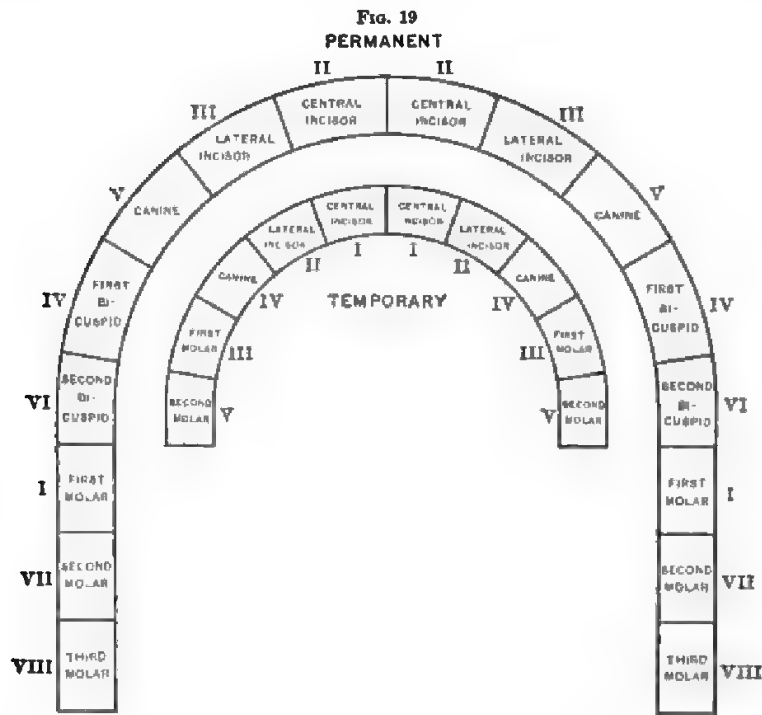


Diagram showing what permanent tooth replaces each temporary tooth, and also the order of succession of the teeth of each set. (Gerrish.)

set to appear are the first molars, popularly called, from the usual period of their eruption, "the six-year-old molars." Thereafter the order of the eruption of the permanent teeth is nearly a repetition of that of the temporary set. On the average they appear about as follows:

ERUPTION OF PERMANENT TEETH.	
First molars . . . . .	6 years.
Central incisors . . . . .	7 "
Lateral incisors . . . . .	8 "
First bicuspids . . . . .	9 "
Second bicuspids . . . . .	10 "
Canines . . . . .	11 "
Second molars . . . . .	12 "
Wisdom teeth (third molars) . . . . .	17 to 25 "



These ages are, of course, subject to some variation.

The process of dentition, especially the first, is often attended by local discomfort and even general disturbance of the digestive and nervous systems. These should not, however, be considered as a normal condition nor neglected as "natural."

**The Saliva.** --The saliva plays no active role in digestion, nor is its quantity considerable until the third or fourth month. Investigators do not agree as to its actual amount at early periods. The amount, however, is manifestly increased at the time mentioned, and very much

FIG. 20



Jaw of a child of seven and a half years, the external table of bone having been cut away to show the stage of second dentition. (Testut.)

so as the beginning of dentition approaches. It is probable that these two developmental facts are ordinarily merely coincidental, as we see pronounced salivation at the usual time even if dentition is greatly delayed. Nevertheless, the flow of the saliva is popularly accepted as the herald of the coming of the teeth and usually it does precede dentition but a short time. When the flow of saliva is well established it continues abundantly, some observers even considering the amount at one year to be equal to that of the adult.

## CHAPTER V.

### GROWTH AND HYGIENE.

HYGIENE has been defined to be "the science and art of the preservation of health." Perhaps this is as good as any short definition, but it involves the presumption that the word health has a definite meaning to the reader. For the purposes of this chapter, hygiene may be taken to mean such rules as to surroundings, conditions, and regimen as conduce to the normal growth and development of a child and the proper functioning of all its organs. Such a meaning necessitates a description of what constitutes the normal state—health, in other words—in infancy and childhood. It should be said at once that this is not a fixed one in any particular, save as we may accustom ourselves to the idea of means and averages. No one questions that breeds of animals widely differing from each other may be equally normal. Racial distinctions in man are usually similarly recognized. In the same environment the tall and the short, the thin and the stout are, within limit, all accepted as normal.

To distinguish between growth and development is not always practicable unless the meaning of the former be restricted to mere increase in height, bulk, and weight, and even these are in part the result of the developmental evolution of organs. If this restricted meaning be adopted, it is safe to say that growth is generally less important than development, and that perfection of function is most important of all. Except for the sake of clearness this matter need not be mentioned, since here, as elsewhere, increase of size without development is merely expansion, not growth.

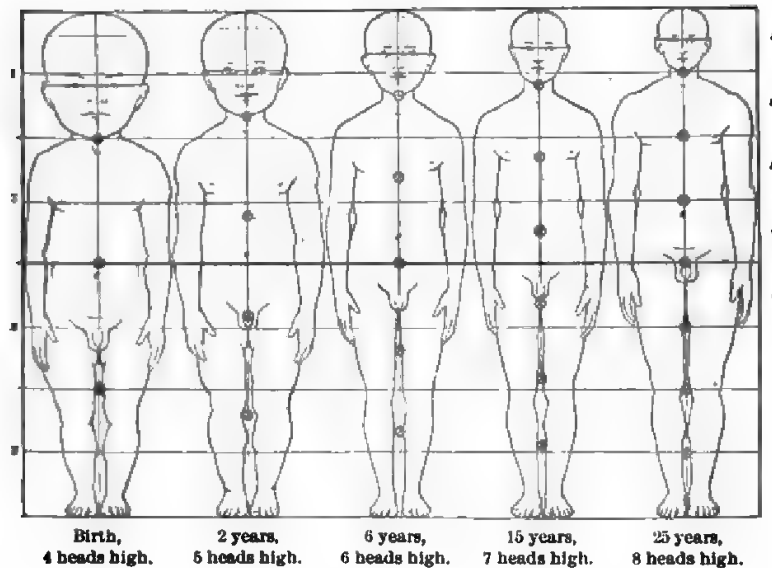
The study of the growth and external development of children from a physiological point of view belongs to recent years. It is scarcely seventy years since Quetelet published the measurements which are generally assumed as our starting point, while the past thirty years—the past twenty in fact—have furnished the greater part of what is now a pretty extensive bibliography. Physiologists, anthropologists, and especially those interested in the application of the physical sciences to the problems of pedagogy, have all contributed to the growing accumulations of observations. But the study of the human form by painters and sculptors is very old. In fact, the old Greek canons have never been superseded. Until these recent years works by artists or upon artistic anatomy were our best, in fact our only, guide as to the proportions of the human form. The artist seeks to establish an ideal or at least to point out the nearest concrete approach to it; while the physiologist and the pædiatrist seek to determine the ways and degrees



in which this ideal may be departed from within the range of normal variations. These artistic studies of proportion are of interest to the pediatricist, to the general practitioner, and to anyone who has the care of the physical development of children. They offer him an ideal toward which to train those he cares for, while the averages and tables of scientific observers give him the knowledge of what are existing facts and a test of his progress toward his standard of perfect success. At the present moment the amount of data gathered by scientific men is very considerable, but more are still needed, and, as in most physical science, the broad understanding of these data awaits an interpreter.

The growth of a child is marked not merely by increase in stature, but by constant change in the proportion of the parts of the body. Discussion of these changes as regards the external figure alone has

FIG. 21

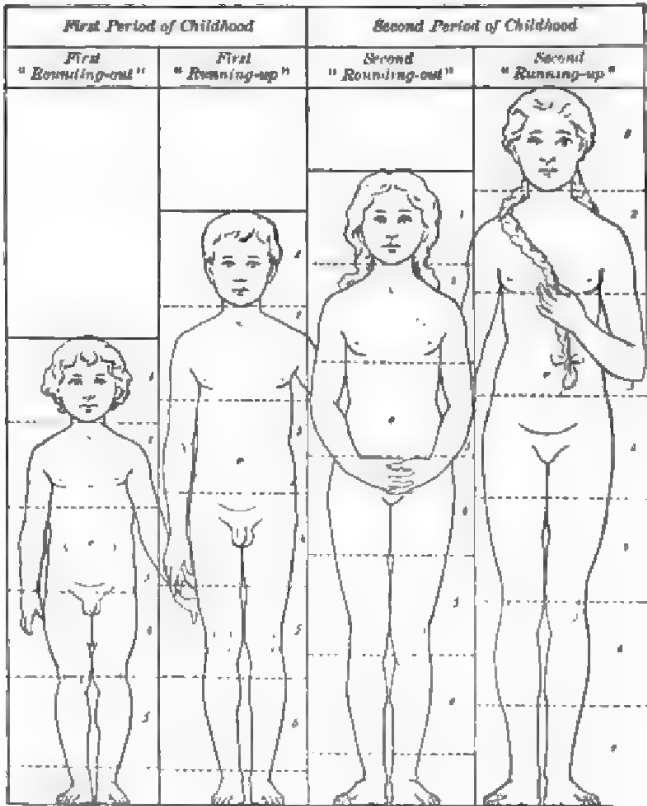


filled volumes, while here but a few paragraphs can be devoted to them. It will save repetition and probably space if the points be first mentioned in which the newborn most noticeably differs from the adult, and then the steps by which they are changed in the course of growth. Diagrams and illustrations will still further save words.

The first diagram (Fig. 21, from Stratz) shows the comparative size of a newborn infant 50 cm. (19.7 in.) long and an adult of 180 cm. (5 ft. 11 in., nearly). It is to be noted that this height is ideal, not the average. The average of upward of 190,000 American-born men in the army during the civil war was very nearly 5 feet 8 inches. The length of the newborn infant is differently given by different observers. Holt found it in some lying-in hospitals of New York 52.07 cm. (20½ inches), about 2½ cm. more than above given.

Differences of proportion as well as of size can be noted, but the former are much more clearly shown in Fig. 22 (also from Stratz, *Der Koerper des Kindes*). It will be noticed that Fig. 21 gives the adult as eight heads high. This is in accordance with the ancient Greek canon and is still accepted as an artistic rule for the male figure. The medical man must, however, not expect to find it holding good except among those whose occupations or amusements give the body every advantage. In other words, it is the canon of the athlete rather

FIG. 22



Normal stages of childhood; outlines from Geyer. The second period nearly corresponds with that of the second dentition.

than of the student or thinker, the latter being more often about seven and a half heads tall. Fig. 21 is divided into eighths by transverse lines, the middle being marked by a heavier line. It will be noticed that the centre of the vertical diameter falls in the adult upon the pubis, rather higher in man than in woman; in the newborn it is rather above the navel, and at intermediate ages at various intermediate points, gradually approximating the adult position. But it may be remarked that many adults present proportions, owing to relatively large heads

and relatively long bodies, not very different from that of the figure given as proper to the age of fifteen years.

The head of the newborn child is two-eighths, or one-fourth, of its height, twice the adult proportion. The trunk is as long as the inferior extremities. In Fig. 21 the five standing figures are drawn as of equal height, which shows the relatively heavy proportions of the infant. The small crossed circles on the vertical diameter of each figure mark the

FIG. 28

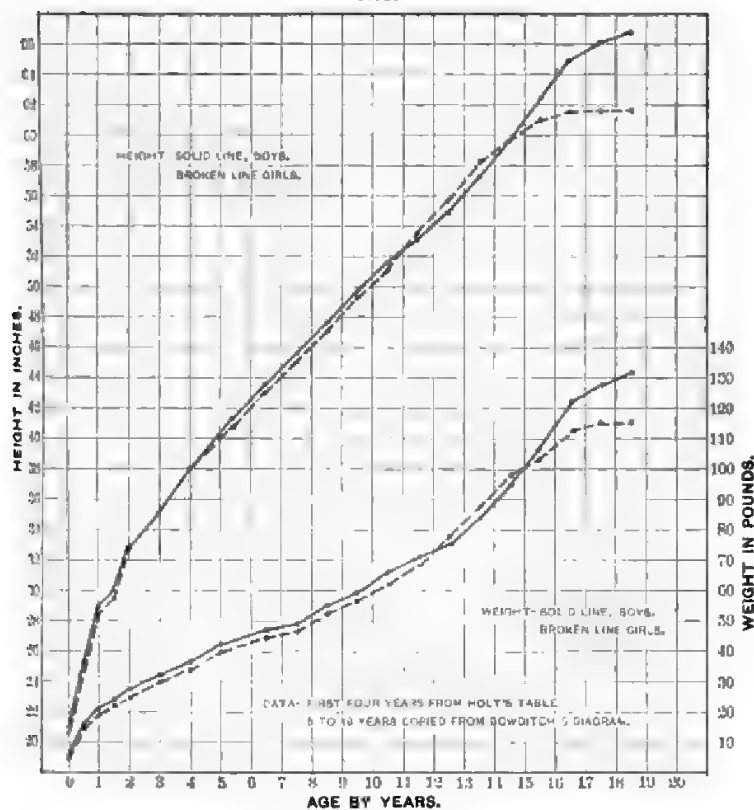


Chart showing height and weight.

head heights for that figure. I have calculated that a man of five feet and ten inches, built on the lines of an average infant, would weigh at least three hundred pounds.

It will also be noted that while the lower extremity is longer than the upper in the adult, the reverse is true in the newborn and gives to the latter a suggestion of simian build. In actual growth from birth to the stature of nearly six feet the height of the head nearly doubles, that of the chest trebles, the length of the upper extremity quadruples, and that of the lower extremity reaches five times its length at birth.

The proportions just given are in round numbers. Vierordt<sup>1</sup> gives the following table as the result of various measurements. The 100 i

FIG. 24

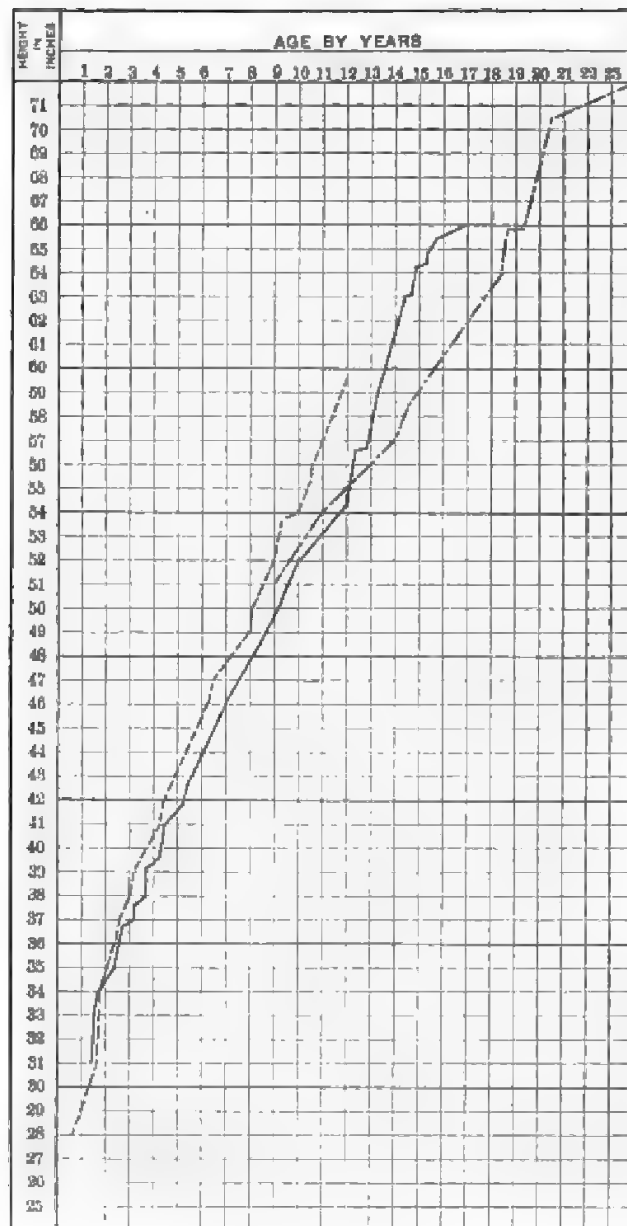


Chart showing height in inches.

<sup>1</sup> Gerhardt's *Kindk*, 1. 82.

the first column represents the average length of that part in the newborn, the other figures represent the relative length or height of that part at the various ages until adult stature is reached.

Part.	Newborn.	End of twenty-first month.	Seven and one-eighth year.	Adult.
Height of head . . . . .	100	150	191.7	200
Cranial part of head . . . . .	100	114	150	157
Face . . . . .	100	200	250	260
Chin to manubrium . . . . .	100	500	700	900
Sternum . . . . .	100	186	300	314
Abdomen . . . . .	100	160	240	260
Inferior extremity . . . . .	100	200	455	472
Height of foot . . . . .	100	150	300	450
Arm . . . . .	100	183	328	350
Forearm . . . . .	100	182	322	350

That this growth is not uniform is well known and its curve when charted presents neither a straight line nor any uniform curve. If the number of observations be large the line will be a wavy one (Fig. 23), but if a single case be chosen and frequent observations be made, the variations may be still more striking (Fig. 24). Similar irregularity exists in the increase in weight, but the curves of the weight line are not identical with those of the stature line, as will presently be pointed out more particularly. It may be mentioned here that these irregularities are more evident the more frequently observations are made. Thus, if measurements be made on rising and retiring it becomes evident that children are taller in the morning than at night, not necessarily that they grow more at night, but rather from the compression of the elastic cartilages by the body weight during the day. On the contrary, the weight is least on rising and increases during the day. But this is doubtless due to fasting, alternating with ingestion of food and drink. More striking, because relieved of these complicating causes, is the observation, made in a considerable number of children in an institution, but which agrees fairly well with my experience, that the periods of increase in stature and in weight are not synchronous. The maximum of growth in stature is between March and the beginning of August, while the maximum increase in weight is between the end of August and December. The maximum of stature increase corresponds with the minimum of weight increase and *vice versa*. So that the progress is alternating. It may be as yet unsafe to take these observations as the basis of definite assertions, but further ones are well worth the making.

Besides these daily and seasonal variations, it is a matter of daily observation that there are longer periods in which the asymmetry of stature and weight increase are pronounced; those periods, namely, of alternate rotundity and slenderness which are called in nursery parlance "rounding out" and "running up," or "weediness," to which attention has been attracted by scientific measurements and tabulation.

If a single case be charted this discrepancy between the curves of stature and weight may be quite striking. In a large number of cases, as the time of the changes vary a little all along the line, the sharpness

of the curves is softened, as in a composite photograph. Nevertheless, as seen in Fig. 24, the general tendency can be noted by the divergence of the line of growth in height from the line of increase in weight. It will be noticed that between, say, five years and eight years the rise in the weight curve is particularly small, while that in the height curve is rapid. The second period of "stretching up" occurs somewhere between eleven and fifteen years. The relatively smaller increase in weight is less well marked in the curves than in the earlier period just mentioned. These alternatives of plumpness and elongation are fairly well represented in Fig. 22.

Returning to Fig. 24, it will be noticed that the lines, solid and broken, do not continue roughly parallel, but cross and recross. In other words, from birth until the twelfth year boys on the average are both taller and heavier than girls. But at the latter age girls pass the boys and continue taller for about three years, while they are heavier than boys for still another year. This period of superiority of stature and weight on the part of girls has been noted, I believe, by all observers, and is, therefore, a universal phenomenon. The relative plumpness and slenderness exhibited in Fig. 22 is expressed scientifically by the proportion of weight to height, a proportion which, of course, must diminish as the frame changes through the stages shown in Fig. 21. But the weight-height ratio for a given height will vary between the sexes, and boys appear to be heavier for a given height up to about 1.473 m. (58 in.), when girls become the heavier. This difference in the ratio is probably not due to differences of fat or development of other tissues, but to the different proportions between the trunk and extremities, which differ in the two sexes and at different ages. Thus for the first ten years the body of boys is longer than that of girls, from ten to sixteen years the body of girls is longer. After fifteen there is little increase in length of the body for girls, while that of boys is considerable, often as much as four inches. Of course, the longer the body in proportion to the total height the greater will be the weight for that height. Similar variations are noticed in the relative length of the lower extremities in the two sexes, these variations being in a general way conversely to those already noted for the body. But after fourteen years of age the lower extremities of girls nearly stop growing, while those of boys gain very much, often as much as four inches.

But Fig. 23 gives not only relative but actual results. From the age of five years to that of eighteen the curves are copied from Bowditch. They represent the results obtained from measurements of 24,595 children in the public schools of Boston, irrespective of nationality. The curve from birth to four years is constructed from figures given by Holt as the result of measurements of his own, covering about 500 observations. Throughout the table, both for height and weight, the solid line stands for boys, the broken line for girls. The average height of a boy or girl of any age can be approximated by noting where the curve crosses the vertical line for that age. The nearest horizontal line followed to the *left* will give the height in spaces of two inches. For

weight take the lower curves in the same way. The horizontal lines followed to the *right* will give the weight in blocks of ten pounds each.

The curves of Bowditch have been selected out of many, not because they are absolute for all children, but because his data were more extensive and more completely worked out than those of any other of nearly equal extent, and the variations from them noted by other observers can be easily alluded to.

Thus if in Bowditch's own tables special classes be compared with the whole, differences at once appear. Children of American parentage show rather greater height and weight (and, according to some observers, greater sitting height, that is to say greater body length as well) than the general average. When observations were made upon the pupils of certain selected schools, such as the Boston Latin School, the Institute of Technology, and some private schools, the height and weight showed a still greater increase above the average of pupils of the same age in the general mass of observations. So, also, the children of non-laboring parents show greater height and weight than those of the laboring classes.

The influence of race, of better nutrition and hygiene in all respects on the side of the "comfortable" classes at once comes to mind, and to a certain degree the influence of these agencies is undoubted. But one cannot go far in the study of recorded observations without finding many other agencies which are operative and noting variations for which the cause is not yet evident. Confining ourselves to statistics taken in our own country, we notice considerable difference in the average of height, for instance, and this difference does not seem to be governed by section or climate, nor to depend upon racial or social distinctions so far as reported. Thus taking the highest average first and ending with the least the order is Pennsylvania; Iowa; New Haven, Conn.; Worcester, Mass.; Oakland, Cal.; Milwaukee; Boston; St. Louis, an order not agreeing with any preconceptions based upon general information. The tallest group in this list is about the equivalent of one year in advance of the last as regards stature. Taking all these groups together, Bos worked out mathematically a table for the "average American" which gives a curve averaging about one-third of an inch higher than that shown in Fig. 23. The practitioner rarely can gather anywhere statistics which will give this "average" value. Drs. Gould and Baxter observed that migration from east to west seemed attended by an increase of average stature. Whether this was due merely to the migration to different climatic and geographical surroundings, as is suggested by the fact that the increased stature of the newcomers is assimilated to that of the earlier residents, or whether improved conditions of living and the fact that in migration, as in early immigration, the migrants are usually in a way picked individuals who live in the open air, contribute to the results is uncertain. Another striking fact was elicited by these observers—namely, that there is a slow increase of stature until the age of thirty-five years.

The most noticeable part of a growth curve, after the sudden rise of



the first two or, perhaps, three or four years, is the rise which marks the puberal or, as it often is, prepuberal growth. The curve in the figure is much less marked for stature than for weight. But, as already said, in both it is rounded off from the fact that this growth is so widely distributed in different cases. Thus we see children, especially girls, who have nearly or quite reached their adult stature at twelve years. (See Fig. 23, lower curve.) On the other hand, it is not rare to find women who have grown materially after marriage, say between eighteen and twenty years of age; and I have by me the chart of a youth who, having been below the average until sixteen, then passed it and between nineteen and twenty-one gained 12.70 cm. (five inches), and still another 5.08 cm. (two inches) after his majority. It is a matter of common observation that the early beginning of this puberal growth is not an indication that the completed stature will be notably great, nor is its delay to be necessarily interpreted in the contrary way. For instance, among my memoranda is one of two sisters whose adult height is the same, but one attained it at twelve years, the other at sixteen. It is interesting to note that however much this growth may be retarded by illness, by adverse circumstances, or by unknown causes, the process is resumed with the greatest pertinacity again and again. So that some observers have been inclined to believe that there is for each individual a certain ultimum of stature, predetermined in some way, perhaps by its own tissue structure, toward which the organism struggles and at which it nearly, if not quite, finally arrives. Such a theory cannot now be proved or disproved, but the student of the laws of growth sees much of encouragement in this persistence of the organism in achievement of a reasonable stature under circumstances seemingly most adverse, even if he must also admit that no one "by taking thought can add one cubit unto his stature."

Space forbids any discussion of the exceptionable stature noted of recent years, especially among women. I have for some years been seeking to find in stock, in habits, in hygiene, and along all the usual avenues of inquiry to find an explanation for the phenomenon, but I have failed to find one at all satisfactory.

Some discussion has arisen over the claim that children who are large for a given age are also forward, as evinced by school grade, for that age. Everyone can call to mind cases showing the contrary in both senses. Yet, while nothing can be predicted in advance regarding any given individual, statistics of large numbers of school children seem to show that there is a general correlation between good bodily nutrition and mental development.

Boas has stated that he found that of children above six years of age the first-born children are both taller and heavier than later children. I have no statistics bearing upon this point during the growing years. But as regards the completed growth of the different children in families my experience is quite the opposite. Possibly different conditions may account for the different findings. Thus, if public-school children are observed, it is possible that they come in a considerable proportion

from families where frequent pregnancies and difficult household conditions may have diminished the mother's vitality *pari passu*. In the "comfortable" classes, whence my observations are drawn, these influences have not been operative, and the improving intelligence of the mother as regards hygienic matters, both for herself and progressively for her children, has doubtless been beneficial to the latter. Besides, it has been asserted as a result of considerable observation that the proportion of the weight of the newborn to that of the mother increases with the number of pregnancies, and there is no evidence that these heavier children do not hold their own with the lighter ones unless the mother's milk fails.

### THE NURSERY.

The nursery should be arranged for before the delivery, and if the accoucheur be the family physician, he can do much by judicious advice to make a hygienic place for the infant. This he is bound to try to do, inasmuch as the susceptibility of a child to the depressing influences of bad hygiene is conversely to its age, the baby suffering more than the child, the child more than the half-grown or adolescent. The nursery, therefore, ought to be the most wholesome room of the house or tenement. Given a fairly healthful home, it is not difficult to make a wholesome nursery. Well-to-do people often indulge in expense or lavishness for the nursery, but often from want of knowledge this goes for luxuries rather than necessities. The necessary expense is not great if it be borne in mind that the essentials are sunshine, pure air, dryness, suitable warmth, and always cleanliness. The first three desiderata are easier gained on an upper floor, but that next the roof is usually too susceptible to external fluctuations of temperature. Even in summer a sunny room is more wholesome than an unsummed one. Morning sun, if it can be had, is preferable to afternoon sun.

**Heating.**—The method of heating the nursery is in towns usually predetermined by the construction of the house. Of the various forms of furnace-made warmth I prefer that of hot water, on the ground that it yields a uniform temperature more easily than other methods. The supply of cold air must be from an uncontaminated source, and the physician would do well to assure himself that this is such. The intake air pipes must be high from the ground, the mouths reversed and screened to prevent things from falling, and animals and insects from crawling into them.

A large amount of moderately heated air is preferable to a small amount of very hot air, because it introduces a larger volume of fresh air and is less provocative of draughts.

The temperature of the nursery is usually too high. If children of various ages must—as is usually the case—occupy the nursery, the requirements of the youngest (and probably the feeblest) have to be taken for a guide. Most American writers set 70° F. as the desired temperature. In England probably 65° F. would be better approved. Probably

70° F. is as moderate a figure as can be hoped for in our usually over-warm, furnace-heated homes, and if children are old enough to go about the house or into other homes, which are sure to be overheated, it is often more prudent to keep the nursery above the ideal heat than to subject the children to frequent variations. I, however, feel sure that healthy children will, other things being equal, be comfortable and will be more likely to remain quite well in a nursery kept steadily at 65° F. than in one at 70° F., and certainly than in one at a higher temperature. If there are very young children in the nursery the night warmth must be kept up. If the situation of the hot-air registers is not already determined, they would better be placed high above the floor, since thus placed they conduce to a better mingling of the air and are out of the way of childish meddling. The open fire is an excellent adjunct to the furnace in severe weather, but it is not desirable in mild weather unless the furnace can be correspondingly controlled.

If there is no furnace the choice of heating mechanism lies between the open fire, Franklins in their various forms, stoves more or less "air-tight," and "heaters." The advantages of the open fire are its cheerfulness and its ventilating value; its disadvantages, its wastefulness of fuel and the unequal warming of the apartment. The Franklins diminish the waste of fuel, and to a less degree, owing to their construction and placing, lessen the inequality of heating. Stoves are economical of fuel, give a large amount of heat, and practically no ventilation of themselves. By "heaters" is meant a sort of stove heating the room in which it is placed and, by means of a register, that above. They have a little of the convenience of a furnace, with the objection that as usually arranged the air sent to the upper room has been drawn from the lower, and is probably already vitiated by the occupants and lights of that lower room. Obviously a nursery should not be supplied with second-hand air. Gas stoves for nursery use need only be mentioned for condemnation. They not only have no ventilating power, but throw their combustion products, not into a chimney, but into the breathable air. This, of course, does not apply to "gas logs" set into fireplaces with flues, but it does equally apply to oil stoves of all sorts burning in the open room and without escape flues.

**Ventilation.** This is the proper place to speak of the effect of lights in vitiating air. A common candle produces nearly as much carbonic acid (carbon dioxide) as an average adult, a large kerosene lamp or gas burner sometimes as much as five or six persons. It is easy to estimate the effect of several lights. If a night light be required it should be as small as practicable and placed if possible beneath a ventilating opening or in the fireplace, so that its combustion gases may escape up the chimney and assist the draught from the room. One thousand feet of *air space* are desirable for an infant, but few rooms can give this amount if an attendant occupies the nursery with it, to say nothing of other

ten. We must accommodate our demands to the possibilities. The best sun-lighted room, already described, must be chosen and the air frequently renewed. While many houses have heating plants, few



have definite ventilating facilities. Probably from convenience the heating apparatus has come in many, perhaps most, houses to do the work of ventilation as well. The open fireplace makes an excellent base ventilator, but it may also make very uncomfortable and even dangerous floor draughts, if the fresh air enters at low levels. This latter point becomes important as soon as children are old enough to play upon the floor, and therefore the air should, if practicable, be admitted at points sufficiently above the floor to permit its admixture with the air of the room which it is intended to purify.

If there is no open fireplace, ventilating flues opening near the floor may be carried up to a convenient height and then into a chimney flue, the draught of which by suction helps to clear out the low-lying foul air of the room. This may be done in several ways, but the principle is one. A ventilating flue to the roof, the vent being capped, solves the same problem. If a stove be the source of heat, the ventilating flue will be more efficient if it be near enough to the stove to have its air column warmed by the latter.

If no flue can be utilized, the various window boards—those with whirligigs, those with elbow tubes, or simple boards—will serve to let in air, and the latter can be contrived to also let out air. Air may enter at the foot of the raised sash, behind the window board, and may enter or leave by the aperture caused by the overlapping of the lower sash upon the upper one. A stout, closely woven cloth may be nailed across the lower part of the window and serve the same purpose as the board. Besides being avenues of ingress and egress for air, windows are great modifiers of the temperature of a room. Here is the formula: Each square foot of glass will chill (or, in hot weather, warm) 1.279 cubic feet of air each minute as many degrees as the difference between the inside and outside temperature. It is easy to figure out the effect upon any particular room. The result is, in cold weather, that the neighborhood of a window is a frigid zone. It is best to place across the front of nursing windows articles of furniture which will keep young children away from this immediate neighborhood.

The ordinary elements of dryness in a house, good drainage, dry cellars, etc., need no comment. Some personal experience leads me to insist that trees near a house are not desirable. They obstruct sunshine and retain moisture. Their function as ornaments and as wind-breakers is best performed if they stand far enough from the house to allow free circulation of air and abundant sunshine.

Cleanliness in the nursery is rather exacting, as the room may at any moment become a sick-bay. It demands a tight, easily cleansed floor, rugs or light carpet squares which can be taken out-of-doors frequently and not swept *in situ*—in other words, the dirt must be removed, not simply stirred up. The walls are better painted than papered, and should be as little encumbered as possible. Window drapery should be very simple; shades and blinds are alone desirable, and stuff hangings are particularly objectionable. Furniture should likewise be chosen with reference to the possibility of keeping it clean. Painted

iron bedsteads and wash-stands seem best to meet the needs. The crib for the baby should be high enough from the floor to escape draught, and it should not be covered with drapery nor placed in a corner of the room. If away from the wall it will be easier to care for the baby and the air will be better. Cupboards and closets, bureaus and wardrobes are constant pitfalls for the household hygienist. Were it not for the terrors of "sweeping day," I would take off the doors from all nursery closets so that everything in them, hanging or on shelves, could be at once seen to be in order, and would be easily and constantly aired. Plumbing in the nursery is another source of untidiness and sometimes of risk. The toilet facilities should be, if in the room at all, the simple bowl and pitcher. Nor should the bath-room and water-closet be immediately connected with nor too nearly adjacent to the nursery. The nursery adviser must keep in mind the welfare of the child, not the ease of the attendant.

If food is to be prepared in the nursery, absolute cleanliness is required in all details, but it will be better to keep all food in an adjoining room. The air of the nursery should never be contaminated by soiled diapers and clothing, nor should the nursery be used as a drying room.

The care of newborn and of premature infants having been treated in Chapter I., we pass here directly to "Nursery Routine."

**Nursery Routine.**—The care of the newborn infant imperceptibly changes into that of the baby. Its baths and clothing are modified or changed as required, but very gradually. In the same manner it advances to the use of its members and to changes of air. These may be spoken of a little in detail.

**Baths.**—The bath, after those necessary for the complete cleansing of the infant after delivery and after the cord is separated, follows usually a simple routine. The immersion bath, of temperature about 100° F., a basin of warm water, bland soap, the necessary towels, napkins and cotton and clothing are arranged in a warm part of the room. The nurse then, having on a thick apron or a bath towel adjusted in its stead, undresses the infant, wraps it for warmth, while she with cotton or cheese-cloth sponges with soap-suds in all the parts likely to be soiled. This completed, the baby is immersed for a moment or two in its bath. This immersion is really for the rapid washing away of the suds by the quickest method, as saving fatigue to the baby as well as labor to the attendant. But it is not at all a necessary part of the bathing, nor should it be made a fetish as it often is. It may be omitted whenever its administration for any reason causes depression or, if an infant be feeble, whenever it be found that other methods are better borne. Ordinarily, a baby when still very young enjoys its bath, and, rejoicing at the freedom of its limbs, turns this nursery duty into a pleasure for the admiring mother or nurse. The bathing of infants and children who are not under direct medical care will be especially mentioned later.

As infancy progresses the temperature of the bath may be slightly lowered. Rules, of course, are not fixed, but only guides. I have been able to devise no better general rule than to drop this temperature,

which started at 100° F., about one degree per month, the bath thermometer, of course, being used.

The further reduction of the temperature of the bath after the first year must also be governed by circumstances. So long as the immersion bath is continued, its temperature must be high enough to ensure no depression and a prompt reaction upon drying and slight friction. By two years of age, if not earlier, the sponge bath may be substituted for the immersion. The temperature may then be considerably lower. If the child stands in water of 90° to 95° F. deep enough to be well above its ankles, thus ensuring the warmth of the extremities, a sponge bath of water twenty degrees below these figures will be well borne by healthy children if it is quickly given, say in a minute. This is the bath of regimen, so to say, making the child better able to meet the day, while the bath for special cleanliness, which is still to be given warm as often as requisite, is best given just before the child is put into bed at night. This same routine may be continued so long as the child has its bath given by another person, the temperature of the sponging water being somewhat lowered and its duration increased according to the completeness and promptness of the child's reaction.

This last phrase suggests the only hygienic contraindication of the bath for infants and children not under medical care. If a bath of any kind at any age causes depression, cold extremities, or a sense of chilliness after the rubbing down, there is evidently an unsuitableness between the child and its bath, the cause of which must be sought out and the disproportion corrected.

**Clothing.**—The essentials of clothing are protection from cold, accomplished without burdensome weight, without constriction or hindrance of the motions of any part of the body or of the extremities. The amount of clothing, of course, must vary with the season for a child who is taken out-of-doors. The nursery temperature being usually fairly fixed and also not very different from the outer air in warm weather, it is better to have the house garments not too burdensome (not so warm, that is to say, as to excite perspiration if the child is active), and to meet lower out-door temperatures by extra garments and wraps. The exact materials and amounts will and must vary with localities, according to temperature and purchasing facilities. Substances of loose texture, such as gauzes, machine-knit or hand-knit material, confine air in their interstices and are better non-conductors of heat than closer woven materials of equal weight. They are also usually yielding, elastic and more comfortable. While by no means advocating any system of undue coddling, I have no sympathy whatever with so-called "hardening" methods, which generally involve the sacrifice of some well-established hygienic principle to a fad or a fashion of dress. I am convinced that uniformity of protection, so far as is consistent with the free use of the limbs, is desirable, and that, for instance, for the prevention of colic warm stockings are needed as well as, if not as much as, warm abdominal covering. Low necks, short sleeves, bare legs have therefore no place in the clothing of young and especially of feeble children.



The clothing of the new baby should follow these general rules: Flexible materials, in easy forms without girdles or waist-bands, arranged to be removed and replaced with the fewest manoeuvres possible. The napkins should be of soft absorbent materials. Linen is traditional, but if new it is hard and stiff, and old linen in sufficient quantity is rarely obtainable. Soft cotton materials, such as stockinet and birdseye, are considerably used, and so far as I have observed are unobjectionable. The napkin should not be needlessly bulky, and pains should be taken to avoid tightly binding the thighs together, while a bunch of material is placed between them. Bending of the femora may result. The napkins for very young infants may be of cheese-cloth and absorbent cotton, and may be burned or destroyed, as they are inexpensive.

While objecting to waist-bands, it should be said that the "band" of the new baby is excepted for the reasons that, save when used as a retaining bandage for the dressings of the cord, it should never be snug enough to exert compression. Its sole function is for warmth to the trunk, the thorax, and abdomen. In infants of ordinarily abundant fat it gives place after a few weeks, or months at most, to the knitted shirt. If it is made tight it is a harmful constriction, and while probably hindering an umbilical or ventral hernia, it probably favors inguinal hernia by furnishing a point of resistance by which unusual and even harmful pressure can be brought to bear upon the inguinal canals if the infant cries or strains very much.

The needless continuance of napkins may be mentioned in connection with clothing. While an infant may need napkins for a year, or for special reasons even longer, there is no doubt that they are usually continued unnecessarily long. If an infant is very early accustomed to have a small nursery vessel placed against its pelvic extremity with regularity it soon associates its presence with the evacuation of the bladder or rectum, and these functions become regular far earlier than would otherwise be the case. The trouble necessary to bring this to pass even in rather difficult cases is certainly much less than that of caring for the napkins soiled during their usually unnecessarily prolonged use.

The need of night napkins is also frequently prolonged by needlessly large or frequent meals of liquid at night. In the section upon Feeding hints regarding the hours for night feeding are given.

One of the most obvious errors in dress and also one of the most difficult to arrest is the cramping of the feet of young children in improperly shaped, especially in too pointed, shoes. Anyone who has taken the trouble to notice the foot of a newborn infant knows that the great toe naturally diverges toward the median line of the body, in a manner comparable to the divergence of the thumb when the hand is pronated, but, of course, in a less degree. A medical man sees at once the folly of distorting this member, but his advice is often disregarded because of the greater ease with which bad shoes are obtained than good ones, and because of the thoughtless vanity of mothers. But good shoes are far less hard to procure than formerly, and it is as well

worth a physician's thought to encourage the use of such as it is to encourage the use of spectacles in proper cases.

**Air and Exercise.**—It is desired that the infant should have the purest air obtainable, and ordinarily out-door air is purer than that of the house. It may, therefore, be taken out-of-doors as soon as it can be properly protected as regards warmth. The conditions are not the same in the country or in small villages as they are in a great city, and what here is said refers to places not crowded. In ordinary summer weather, when the house is not artificially heated and windows are kept open much of the time, the infant may be taken out very early, as soon as its need of especial warmth is passed. This is practically when it has regained the initial loss in weight of the first few days, say when it is a week or ten days old. It is, of course, to have additional wraps if the out-door temperature is lower than that of the house. In this warm season, when windows are open, there is less gain as to purity of air by going out-of-doors than at other times. The child should be accustomed to out-door airing even then, as establishing the out-door habit before cooler weather arrives. Even in summer the child should be protected from the wind and its eyes shielded from strong sunshine.

If the birth has occurred in the spring or autumn, rather more management and circumspection are needed, and this is increasingly true if the baby comes in winter, when it is rare that a baby can be judiciously taken out-of-doors under the age of a month, and this only in moderate weather. In severe winters it is sometimes very hard to find suitable days for a very young baby to be taken abroad. The warm part of the day must be chosen, and sunny, sheltered nooks be sought for. I believe also that it is safer to take quite young children in the arms, not in the baby carriages. They thus get the warmth and support of the attendant's body, and the child's wraps must be arranged not for display of embroidery, but for the best protection of its body and extremities. The airing must be brief, say a quarter of an hour at first, and this time prolonged gradually as its effect is noted. If a baby has gotten a good start in the warm season it is easy to keep it out daily an hour or two as the cooler season advances, with the protection of the baby carriage, wraps, and foot-warmers. Thus safeguarded they may get the air or even sleep out-of-doors without harm.

In great towns, save for those living very near to parks or open spaces, the problem of airing is less simple. One who walks the main streets of a residence district sees a good many objectionable features as to the airing of babies, and more especially of young children. The baby carriages are often massed in great numbers, proceeding more in accordance with the conversational fancies of the nursery maids than with regard to the protection of the children's eyes from sun or their air passages from dust and dirt. In fact, this latter is often impracticable when upon every block buildings are coming down or going up and the street dirt of all sorts is blown about by the wind forced along narrow streets flanked with high buildings. The older children who are out in carriages, but who solemnly walk along beside the carriages, are

even less well protected. In cities which are not well and constantly cleaned the untidy gutters still further deteriorate the air.

Under such circumstances, many years ago, I adopted the plan of in-door airings; that is to say, of opening widely the windows of a sunny room as high up as practicable, so as to avoid as far as possible the gutter and sewer emanations, and let the children, dressed for the street, play in this room, the windows being adjusted, while the children are in it, so that they shall not be improperly placed in draughts.

Infants in ordinary health will get all the necessary exercise if their limbs are free from restraint, kicking, rolling about and, as they gain the use of limbs, in grasping objects, in creeping, and finally in walking. So, later, as young children, they will get in play all the necessary muscular exercise; and if this can be taken in free air, so much the better. One of the advantages of the plan of in-door airing, just described, is that children who are too young to play upon damp or icy ground may play in a cold, well-aired room. If the airing were out-of-doors the child would of necessity be in a baby carriage.

In the country the question of exercise scarcely arises for children who are not ill. In towns after school life is begun, and especially for girls, the problems of out-of-door exercise is not always an easy one to solve, owing to the lack of interest in their out-of-door surroundings to very many children. Walking in streets is dreary, park facilities are not always convenient, and monotonous if at hand. The best solution in great towns or cities is often the clubbing together of a number of families to hire, as a leader in sports and exercise, an intelligent young person, male or female, who can interest the younger ones in active play beyond the power of the ordinary nursery attendant.

**Sleep.**—A newly born healthy infant sleeps so large a part of the time that its existence might be described as sleep interrupted with intervals of feeding, to which civilization adds the disturbances of the toilet. It follows that if the food be proper in kind and amount and the sleep ample, healthy development is likely to follow. The importance of proper food for the infant is pretty well and generally understood, however poorly its furnishing is carried out; but the necessity of good habits of sleep, except so far as the comfort of the baby's attendant is concerned, is far less appreciated. During the first week the infant may prefer to sleep to taking food, and unless it be actually hungry it may continue to sleep when put to the breast. The need of food presently increases, however, and as it develops it remains awake more. The actual number of waking hours of a healthy and comfortable infant will, of course, vary with individuals, probably from six to eight in a day by the time it is six months old; nor will it take much less sleep at the end of a year. The more active, bodily and mentally, a child is the more hours it is likely to desire to remain awake. But for this very reason it needs more rest, and it is for these active children—good health being assumed—that good sleeping hygiene is most imperative. So long as an infant receives food, natural or artificial, during the hours ordinarily devoted to sleep by an adult, we cannot easily divide the day's sleep

and the night sleep. When, however, the night feedings are reduced to one or to none the routine is easier to enforce. This date should be when the child is about six months old. (See section on Infant Feeding.) But the physician must see that a good regimen of sleep is already well established, or he will be likely to experience great difficulty when night feeding is abandoned.

As has been already said, up to six months of age the amount of sleep is large, the hours of feeding, of a little play or attention to its surroundings and of its toilet being about all that it spends awake. After the night feeding is abandoned, the evening sleep until the feeding at the mother's bedtime and the night sleep together will make about twelve hours. In addition there will be two daily naps, say one of two hours in the latter part of the forenoon and another one of an hour or more in the afternoon, and this routine is likely to continue during the first year and it should be continued as long as practicable. But during the second year, while night sleep continues little abbreviated, the second nap is usually not obtained, the whole day's sleep being perhaps no more than fourteen hours. The day's nap should be continued just as long as possible, and if a child is no longer able to sleep in the daytime a period of rest in the crib with the shoes removed and the dress loosened, if not removed, is of great advantage, especially as a restorative to the nervous system. The amount of sleep required as children increase in age gradually diminishes, but until the growth is quite complete they should have a larger amount than an adult, and it is better that they should take all that the pressing demands of school life allow. Even after twelve years of age ten hours of sleep should be aimed at, nor should this amount be much curtailed before sixteen.

Good habits of sleeping are often already established by the monthly nurse if she be judicious. The conditions conducive to quiet sleep are comfort of body and quiet surroundings. The child must be free from constriction or irritation from its clothing, must be thoroughly warm, but not burdened nor overheated by its coverings, should have a comfortably full but not overdistended stomach, and its bowels not constipated. If, then, it be placed in its bed in a quiet and dimly lighted room it is pretty certain to sleep unless it has been taught some disturbing habit. If it has been put to sleep by rocking, by holding in arms or by any similar method it will not always relish being laid down. If it is put to sleep in a stuffy, overheated apartment, or in one (pity that it need be mentioned) filled with the smoke of the admiring father's pipe or cigar, it can hardly be expected to rest well. Nor if it has been dandled and played with until it has become excited can it compose its exalted nerves at once. Whenever one familiar with infants hears of restlessness at night he pretty certainly inquires concerning the night meal. If the infant be upon the breast, he will suspect a failing milk supply. Many such an infant is promptly cured by a bottle of proper food. On the other hand, with bottle-fed children the first inquiry is regarding an excess of food. The stopping of the last bottle and the readjustment of the day's routine of feeding often brings quick relief.



## SECTION III.

# INFANT FEEDING.

By THOMAS S. SOUTHWORTH, M.D.

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### CHAPTER VI.

#### MATERNAL FEEDING—WEANING.

#### **MATERNAL FEEDING.**

THE milk of each mammalian species is especially adapted to the needs of its young. It is so constituted as not only to nourish and furnish the requisite elements for growth, but, by a delicate adaptation to the digestive organs of the young, to gradually develop these for the task of digesting the kinds of food upon which it will subsequently subsist. As brought to the attention of the profession by Chapin, biology furnishes us with incontrovertible evidence of these facts. In certain mammals, such as the kangaroo and the American opossum, the mouth of the fetus is directly adherent to the teat. In an especially constructed pouch the nourishment and growth are effected in this manner without any placental connection whatever. In these mammals there is both anatomical connection and physiological dependence on the mother. In placenta-forming mammals the anatomical connection of the young is severed at birth, but the physiological dependence upon the mother remains and continues until further development of the organs of locomotion and digestion fit it for independent existence. The attainment of this independence is deferred much later in the human species than in any other; and since in man the digestive and nervous systems are notably undeveloped at birth, it is to be expected that the secretion of the mother's breast is presumably adapted for these special needs.

The milks of all species of mammals have certain characteristics in common, in that they all contain fat, sugar, proteids, mineral salts, and water. While fat is necessary for the proper formation of the osseous and nervous systems, and sugar is capable of being transformed and stored up in the body as fat, both of these have more ordinary uses. It may be said in general that fat and sugar are the heat and energy



producing elements which keep the young alive and furnish the motive power to the body, while proteids, which alone contain nitrogen, are the real constructive elements which build the body, making blood, repairing waste, and forming new cells in growth. Fat and sugar are, for practical purposes, much the same in the milks of all species, but this is not the case with the proteids. The young of the different species differ greatly in the rapidity of their growth and the length of time after birth in which they mature and become independent of the mothers' mammary glands for nutrition. This readily explains the necessity for decided differences in the amount of the tissue-building proteids in the milks of the different species.

#### MILK PROTEIDS.

The proteids of milk are divisible into casein and a group of soluble albuminous bodies formerly classed as albumins. Casein is precipitated in more or less solid form by the action of acids alone or in the stomach during the process of digestion. The soluble albuminous bodies are not precipitated by acids or during digestion, but remain in the fluid which separates from the casein and are readily absorbed by the digestive tract. Moreover, the physical characteristics of the casein curd, formed in digestion, whether small or large, soft or tough, bear a definite relation to the type of food consumed by the adult of the species and its digestive organs. This latter principle, that the stomachs and digestive tracts of different mammals vary in construction and in their proportions according to the kind of food upon which the adult individuals must live, is well established by comparative anatomy. The carnivorous animals feed upon concentrated food, and have small digestive tracts. The herbivorous animals, on the contrary, consume bulky food in which the proportion of nutritious matter is comparatively small. They have roomy digestive organs, which functionate best when distended.

Taking the most important examples for our purposes to show the adaptation of the mother's milk to her young: cows' milk coagulates in large masses in the calf's stomach, filling the organ and thus developing the digestive capacity against the time when it shall be required. The human infant has a very different type of stomach. It requires preparation for a less bulky food, and its smaller stomach receives milk which is coagulated in small, soft flocculi. But this difference in the way the casein coagulates in the milks of the species is not solely one of the quantity of casein contained in each milk, although the amount is considerably greater in cows' milk than in breast milk, else when cows' milk is proportionately diluted or modified to resemble breast milk the physical characteristics of the curds should be the same.

This is, however, not the case, for the curds found in such modified or diluted cows' milk are larger and tougher, and the conclusion is forced upon us, although not as yet absolutely proven, that there are distinct differences in the caseins of the various mammalian milks.

These caseins seem to be dissimilar bodies which react to rennet,

acids, or the digestive juices in very different ways, and it is definitely accepted that they are not interchangeable with equal digestibility for the stomachs of the young of different species.

Important as the question is, our knowledge of the chemical changes taking place in casein when subjected to the action of acids, as during digestion, has been based upon a false theory, owing to the general acceptance of the incorrect deductions drawn by Hammarsten from his experiments. He wrongly concluded that there was no ground for believing that any chemical combination takes place between the casein and the acid used to precipitate it.

The recent epoch-making discoveries of Van Slyke and Hart<sup>1</sup> completely disprove this, and show clearly that the acid combines directly with the casein, forming a definite chemical compound.

The possession of such a clear conception of the processes taking place during the earlier stages of the digestion of milk has long been awaited, and will prove invaluable in comprehending the hitherto obscure and complex problems of infant feeding.

When the young animal is born the mammary glands secrete colostrum, which is gradually transformed into true milk. Just as in the lower orders of animal life the stomach is a later and specialized development of a part of the intestinal tube, the digestion of the newborn is intestinal until the functions of the stomach are developed. Colostrum is less readily coagulated than milk, and, being suited for intestinal digestion, passes quickly through the stomach, but in its passage has the effect of awakening and stimulating the digestive secretions of the stomach.

**Digestion of Casein.**—Casein occurs in milk combined with calcium, and is now known as calcium casein. The earliest secretion of the young stomach is the enzyme rennet. This ferment, acting upon the calcium casein of milk, forms a soft clot known as calcium paracasein (junket). Until acid is secreted this calcium paracasein clot may pass on into the intestine, where it is readily digested by the pancreatic and intestinal secretions. In the absence of acid, pepsin cannot attack calcium paracasein. But when the stomach begins to secrete hydrochloric acid in small quantities the acid combines with the calcium of the calcium paracasein clot, releasing free paracasein (a base-free proteid) which forms a firmer curd. This curd of free paracasein is now readily attacked by pepsin, and true stomach digestion is inaugurated.

This free paracasein presents new physical characteristics, forming a curd firmer than the soft calcium paracasein clot, and having a tendency to shrink. It is soluble in a dilute solution of common salt, is readily digested by pepsin, and is probably almost exclusively formed during the period when the young stomach secretes but a small amount of weak hydrochloric acid, only sufficient to combine with and remove the calcium from those parts of the paracasein clot with which it comes in contact. The remaining unaltered paracasein still passes on into the intestine to undergo digestion. As the acid secreted by the stomach

<sup>1</sup> Bulletin 261, the New York Agricultural Station, Geneva, N. Y., January, 1905.

increases to a point slightly beyond that necessary to combine with the calcium of the portions of the paracasein with which it can come in contact, the excess of acid is used up in combining directly with the most exposed parts of the free paracasein, forming hydrochloride of paracasein a definite compound of proteid with acid. This is tougher than the free paracasein, differs from the latter in not being soluble in dilute salt solution, has a similar or greater tendency to shrink, and, as long as the hydrochloric acid secreted is completely used up in forming the products mentioned, is not so easily or rapidly digested by pepsin as the free paracasein. It therefore tends to stay longer in the stomach and to prolong gastric digestion.

But since the paracasein clot is attacked upon its surface by acid, and curds, especially of the milks of different species, may vary much in size and density, the chemical action of the acid may penetrate them to different degrees, and it is consequently entirely possible to have at the same time, within the curd or in the gastric contents in varying proportions, paracasein hydrochloride, free paracasein, and calcium paracasein depending either upon the admixture or contact of the acid with the stomach's contents or upon the strength and quantity of its gastric secretions.

As the stomach becomes able to secrete more acid, more of the paracasein is changed into free paracasein and the acid salt of paracasein; more of the milk then remains in the stomach prepared for gastric digestion, and this stimulates more secretion of hydrochloric acid and pepsin. When, finally, during the process of digestion more acid is secreted than can combine with and saturate the exposed portions of the paracasein so that free acid is present, pepsin digests the hydrochloride of paracasein with greater facility. Although digestion progresses more rapidly when free acid is present, this is now counterbalanced by the large quantity of material requiring stomachic digestion.

We are, therefore, in a position to grasp one of the most remarkable phenomena in nature, namely, that milk which itself retains, after the end of the colostrum period, practically the same composition throughout lactation, is changed by the action upon it of the developing and increasing gastric secretions of the young into forms and compounds which at first require moderate, and later, more extended gastric digestion, by which means the stomach is progressively called upon to perform more and more work, until it is sufficiently developed anatomically and physiologically for the animal to begin its subsistence upon the types of food consumed by the adult of its species.

Although this automatic adjustment of the milk of the mother to the digestive secretions of her young is under normal conditions practically perfect, there are marked differences in the form and density of the curds formed from the caseins of different milks, so that the use of the milk of another species may readily cause difficulty or disturbance. The time then has passed for considering milks of different species to be practically the same because of gross resemblances. Human milk is especially designed for the human infant and cannot be exactly

imitated from the milk of the cow or any other animal, much less in the laboratory of the manufacturer of infant foods.

Nature intended that the human infant should be nursed at the maternal breast after birth just as much as that it should be nourished by the placental blood before birth. The secretion of the breast is designed not alone to support life, nor only to furnish material for ordinary growth, but physiologically to complete the development of those organs which are but partially developed at birth.

#### THE SECRETION OF THE HUMAN BREASTS.

**Colostrum.**—The early secretion of the breasts after the birth of the child is distinctly different from that which is later established. It is less sweet, of a yellow color, scanty in amount, less readily coagulated, acting as a stimulant to the digestive organs and containing microscopically, besides fat globules of unequal size, certain cells called colostrum corpuscles. These have a small, degenerated nucleus, a granular protoplasm, and are considerably larger than the fat globules. Their persistence in the gradually changing secretion marks the duration of the colostrum period. It is most distinctive during the first two to three days when the secretion is small, the color deeper, and the corpuscles more numerous. The proteid percentage is increased in proportion to the presence of the corpuscles, which normally disappear in from seven to twelve days after birth. Persistence beyond this period or recurrence of the corpuscles later in the milk is abnormal and liable to cause disturbance of the infant's digestion. For this reason, except for newborn babies, a wet-nurse should have passed the colostrum period. Where there is persistent digestive disturbance at the mother's breast immediately after birth, other nourishment may be given and the breast pumped until after this period is passed, when nursing may be usually resumed.

**Breast Milk.**—Breast milk is an opaque, bluish-white, rather sweet fluid. Its reaction is usually stated to be amphoteric, but with phenolphthalein, a much more sensitive indicator than litmus, it has been shown by Kerley, Gieschen, and Myers to be faintly acid. The average specific gravity is 1031, with variation from 1028 to 1034. The addition of weak acid causes a moderate coagulation in fine, soft flocculi. The fat globules under the microscope are approximately of the same size.

After the colostrum period, with its low sugar and higher proteids and salts, is passed the composition of breast milk, when uninfluenced by ill health or faulty hygiene, becomes fairly uniform. Adriance, however, has shown that while the sugar tends to rise very slightly, the proteids and salts toward the end of lactation show a moderate descending curve, which is doubtless one of the factors in the causation of those cases of rachitis which result from unduly prolonged nursing. The composition of milk will be more fully discussed in the chapter on "Cows' Milk."

Experience teaches that the maintenance of certain normal relations

between the percentages of fat and proteids are of importance for normal digestion and proper nutrition.

Diarrhea and poor digestion occur with excessive fat, indigestion with too high proteids, and poor nutrition with deficient proteids or fat. Decrease in the latter causes a tendency to constipation.

Nitrogen being necessary for tissue building, the proteids which contain the nitrogen of the milk become perhaps the most important element. The soluble proteids which remain in solution are usually stated to exceed in amount the casein which is precipitated during digestion, and this constitutes but one of the important differences between woman's and cows' milk. In the latter the greater actual as well as proportionate amount of casein, which is also of a different character from that of breast milk and coagulates in larger and firmer masses, renders it more difficult of digestion. But the mere enumeration of these elements, fat, lactose (milk-sugar), soluble proteids, casein, and salts, whose amounts can be estimated, does not probably reveal some of the most vital elements, namely—the ferments and protective principles which adapt breast milk to the infant's digestion, and which, as shown by Roger and others, render the nursing infant largely immune to the infectious diseases.

**Automatic Adjustment of Breast Milk to the Stomach Secretions.**—The stomach of the infant at birth is but slightly developed both in size and power of secretion. The first secretion of the breasts, colostrum, is rich in soluble proteids, which require little if any action by the stomach before they can be absorbed by the intestines, into which they are quickly passed; but they also have the property of stimulating the hitherto unused functions of secretion and absorption, so that during the gradual change to the more permanent breast milk the stomach is gently initiated into its new duties.

The rennet ferment secreted by the stomach acts upon the casein, forming a flocculent precipitate which tends to remain longer in the stomach. Hydrochloric acid is soon secreted, which acts upon this, forming a soft, finely divided curd, and pepsin is secreted to digest it. The stomach thus takes upon itself more and more of the work of digestion, increasing the complexity of the products formed, the firmness of the masses, and the time required for the completion of stomach digestion. This furnishes a rational explanation for the need of lengthening the interval between nursings, and, when cows' milk is the food, of allowing sufficient time for the last meal to leave the stomach, since cows' milk forms firmer masses and requires longer digestion. Thus we see that while breast milk after the end of the colostrum period does not materially change during lactation, the stomach elaborates from this unchanging supply of raw material new compounds which make new demands upon the development of that organ, until it is at last fitted to begin the digestion of other forms of food at the time of weaning.

**The Chemical Composition of Breast Milk.**—This has no hard-and-fast lines in percentages. Averages obtained by combining the results of many examinations are useful in a schematic way, but in nowise inform

us of the variations which take place in the milk of different women, and even in the milk of the same woman on different days and under different circumstances; these are influenced by the length of interval between nursings, by her health, diet, and the condition of her nervous system. Holt gives the following table:

COMPOSITION OF BREAST MILK (HOLT).			
	Average per cent.	Common healthy variations per cent	
Fat . . . . .	4.00	3.00	to 5.00
Sugar . . . . .	7.00	6.00	" 7.00
Proteids . . . . .	1.50	1.00	" 2.25
Salts . . . . .	0.20	0.18	" 0.25
Water . . . . .	87.80	89.82	" 85.50
	100.00	100.00	100.00

It should be borne in mind, however, that considerable variations from this average exist in the milk of many mothers whose children are digesting perfectly. In a series of analyses from 14 healthy breasts, upon which infants were thriving, Harrington found the proteids to vary from 1.08 to 4.17 per cent. Only 4 were below 2 per cent., 5 exceeded 3.50 per cent., and of these 2 were somewhat over 4 per cent. The fat varied from 2 to 5 per cent. From this we must conclude that the usual averages laid down for the fat and proteid of breast milk are no criterion of the digestive powers of the individual infant. They have much the same variability that we find in infants for the digestion of the fat and proteids of cows' milk, although the latter are more difficult of digestion, which to some extent explains the failure of attempts to feed all children artificially upon modifications of cows' milk based rigidly on these averages. On the other hand, when a chemical analysis shows a decided variation from these accepted averages, with disturbed digestion in the infant, especially if there be high proteids, we are in possession of valuable data upon which to base our tentative treatment of the mother to remove these probable causes of the disturbance.

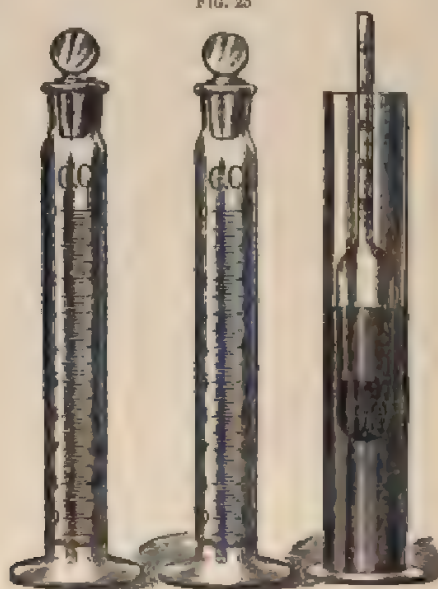
**The Clinical Examination of Breast Milk.**—When proximity to a well-equipped laboratory and the circumstances of the patient allow, the complete chemical analysis of the milk gives us the most accurate and valuable information, but the inability to secure such analysis should not lead the practitioner to neglect the simpler methods of gaining an approximate knowledge of the quality of a given milk, which he may himself carry out with very little time or expenditure. Most important for our purpose is a knowledge of the specific gravity and of the percentages of fat and proteids in any case where the quality of the milk is questioned, to which may be added, if desired, a microscopic examination. The milk-sugar, as we have seen, varies very little and may be disregarded. The salts or mineral matter, although they vary, we have as yet no known method of influencing, and are of minor importance. Since in many cases calling for examination only small quantities can be obtained with the breast pump, our apparatus must be small enough to utilize this. Milk for any test should be taken from about the middle of



the nursing, when the breast is about half-full, to obtain average milk and to avoid the thinner first milk and richer last milk. About one-half ounce is required. The specific gravity may be taken with any small urinometer.

*Determination of Fat.*—For the estimation of the fat small tubes are now made and sold which can be used in the centrifuge apparatus now employed by most physicians, and which are upon the same principle as the larger tubes of the Babcock machine used for that purpose in the modern dairy. This latter can be used, but requires at least 17.50 c.c. (5ss) to determine the fat. A simpler, less accurate, but still valuable apparatus made by Eimer & Amend, of New York, and costing \$2, is the Holt Apparatus for the Clinical Examination of Breast Milk (Fig. 25),

FIG. 25



Holt's apparatus

which consists of a small lactometer (hydrometer) and two tubes containing 10 c.c. each, graduated in hundredths, one of which is filled with milk exactly to the 100 mark and allowed to stand twenty-four hours, after which the amount of cream which has risen may be read upon the scale. Five per cent. of cream is equal to 3 per cent. of fat in the milk. For emergency purposes this may be roughly imitated by using a narrow, flat-bottomed test-tube and a centimetre scale.

*Determination of Proteids.*—Unfortunately the exact percentage of proteids can only be determined by elaborate chemical analysis, but an approximate estimate can be made from the specific gravity and the cream percentage. This depends upon the principle that fat being lighter than water and proteids heavier, a high cream content lowers the specific gravity and a low cream content raises the specific gravity.

Therefore, a high specific gravity with high cream indicates excessive proteids, and a low specific gravity with low cream deficient proteids. Deductions from the findings in any given case may best be made according to the following table, which accompanies the Holt apparatus:

WOMAN'S MILK.			
	Specific gravity, 70° F.	Cream in 24 hours.	Proteids (calculated).
Average	1.031	7 per cent.	1.5 per cent.
Normal variations	1.028-1.029	8 per cent. to 12 per cent.	Normal (rich milk).
Normal variations	1.032	5 per cent. to 6 per cent.	Normal (fair milk).
Abnormal variations	Low (below 1.028).	High (above 10 per cent.).	Normal or slightly below
Abnormal variations	Low (below 1.028).	Low (below 5 per cent.).	Very low (very poor milk).
Abnormal variations	High (above 1.032).	High.	Very high (very rich milk).
Abnormal variations	High (above 1.032).	Low.	Normal (or nearly so).

**Microscopic Examination.**—This is of no value in determining the richness of fat, but may be employed to search for colostrum corpuscles, pus, red blood cells, or by staining to show the presence of micro-organisms.

MANAGEMENT OF LACTATION.

It is quite as often with ignorance of the proper rules to be followed during lactation as with the correction of abnormalities in breast milk that the practitioner is called upon to deal, and the management of the nursing mother and her child must be considered largely from this standpoint.

The correct management of lactation begins at birth with respect both to diet of the mother and the nursing of the infant. The infant should be placed at the breast as soon as the mother is somewhat rested, and for the following twenty-four hours it should be allowed to nurse every six hours. The second day an interval of four hours is observed, and with the establishment of the flow on the third day the infant should nurse every two hours. The following table gives this succinctly:

NURSING SCHEDULE.			
Age.	Interval through day (hours).	Number of nursings in 24 hours.	Night nursings between 9-10 P.M. and 6-7 A.M.
During first day . . . . .	6	4	1
" second day . . . . .	4	6	2
" third to twenty-eighth day, inclusive . . . . .	2	10	2
" second to third months, inclusive . . . . .	2½	8	1
" fourth to fifth months . . . . .	3	7	1
" fifth to eleventh months . . . . .	3	6	0

Frequent and prolonged tugging at the breast, when the secretion is scanty during the first two or three days, only serves to increase the danger of producing excoriations of the nipples. In the interval the infant should be given three or four teaspoonfuls of plain boiled water every two hours, which stimulates the circulation of the stomach mucosa, increases the urinary flow, washing out from the urinary tubules the urates whose presence in concentrated urine leads to much unexplained discomfort and crying of the newborn. Infants given water during this

period show less initial loss of weight. Night nursings should be strictly limited according to the schedule, in the interest of both mother and child. After nursing the baby should be laid down to inaugurate good habits at the start. If the baby is feeble or puny at birth, a 5 per cent. solution of milk-sugar may be substituted for plain water. If the flow of milk does not begin on the third day, especially if the infant shows the so-called inanition temperature, which may reach 103° F. or over and is due to lack of food, a low formula—fat 1.00, sugar 5.00, proteids 0.33 (see page 146)—should be given, replacing each alternate two-hourly nursing, and will be followed by a drop in the temperature.

#### DIET OF THE NURSING MOTHER.

The diet of the nursing mother should at all times include abundant fluids in order that the secretion of the breasts may be carried on without encroaching upon other fluids required for normal functions. The amount demanded is much larger than that furnished by ordinary diet and should not be left to the inclination of the patient, but extra fluids should be given at stated intervals. This should be definitely impressed upon the patient, nurse, or attendant friends. The administration of fluids should be begun as soon after childbirth as the stomach will retain them. They add to the mother's comfort and flush the body through the kidneys of much effete matter which may otherwise be excreted in part by the breasts, disturbing the child. The urine of the mother during the first few days of the parturient period is usually dark and concentrated, owing to the decreased intake of fluids during parturition, loss of blood, and the rapid tissue changes in the period of readjustment. On the first day give at frequent intervals water and nutritious fluids, such as milk and gruels, or, if necessary, mutton-broth or chicken-broth. On the second day, nutritious fluids and simple semisolid food. On the third day, in an uncomplicated case, digestible solid food may be added. This may usually include a small amount of meat, once a day, if the child is having no digestive disturbance due to too high fat or proteids which the ingestion of meat by the mother may increase or maintain. In general, during lactation, the mother should eat abundantly of those simple, nutritious articles of food which she knows by experience she can eat and digest without difficulty. Fewer special articles are to-day tabooed than formerly. Milk, eggs, meat, cereals, fruits, and vegetables allow a sufficient range. Certain of the stronger vegetables must be avoided by some mothers, while others take them with impunity. Tea and coffee should be partaken of sparingly if at all. They are stimulants which have no milk-making properties, and the former especially may disturb the mother's digestion and its alkaloids affect the child through the milk. Beer has little nutritive value, and this and other alcoholics often disturb the infant. Malt extracts have more value and are chiefly useful to increase the fat in the milk, although at times they also increase the flow. Besides plain water I recommend:

1. **Milk.**—Of this at least one quart should be drunk daily. No argument seems necessary to show that this furnishes in the simplest and most readily assimilable form the materials needed for milk secretion. In cases of faulty digestion it may be diluted, heated, peptonized, or given as zoolak, or kumyss.

2. **Cornmeal-gruel.**—While not necessarily required by those mothers who naturally have an abundant flow of milk, experience has shown that this has no equal in restoring a deficient secretion. It should be cooked at least four hours in a double boiler (Fig. 26) and well salted to taste. When used, it should be thinned with water or milk so that it can be drunk, not eaten with a spoon. Two or three bowlfuls should be taken in the twenty-four hours. If there be any difficulty in digesting it, it may be dextrinized, which both thins it and allows the use of more cornmeal in each portion, or, if need be, furnishes more nutritious material with smaller bulk. Such dextrinized gruel is made as follows:



FIG. 26

Double fernus boiler.

Take three-fourths teacupful yellow cornmeal, one quart cold water, two teaspoonfuls cereal, and sufficient salt to flavor. Mix in a double boiler; bring slowly to a boil to allow the dextrinizing agent to act, and cook for two or three hours. It may be strained and taken plain or mixed with equal parts of milk if preferred.

3. **Cocoa.**—Some mothers can digest as a hot beverage, in place of tea or coffee, the so-called "cocoas," which are apparently only chocolates from which a part of the oil has been removed. Better usually than these, and without effect upon the digestion, is the cracked cocoa bean, which, after prolonged boiling, loses its first slightly bitter taste and makes an agreeable, nutritious beverage. The pot in which it is cooked should be kept simmering on the back of the range, and water added from time to time. It should be emptied but once a week, and a small quantity of the cracked bean added daily.

#### HYGIENE OF THE NURSING MOTHER.

**Postpuerperal Anemia.**—Most women are anemic after childbirth and this condition is usually neglected. Proper digestion and secretion is dependent upon the quality of the blood. Bland's pills are a satisfactory form of iron.

**Constipation.**—Constipation is not compatible with good health, and unless relieved effete material may be thrown off in the milk. Cascara in some form is best adapted for routine use. Salines are contra-indicated in nursing mothers, as they reduce the flow of milk.

**Exercise.**—Daily exercise, either by driving or, far preferably, by walking should be begun at the earliest moment that the mother's



condition allows. These walks should extend, if possible, to one to two miles daily, but should always stop short of actual fatigue.

I have long taken the position that in the vast majority of cases the milk of a healthy mother who takes sufficient out-of-door exercise and eats sensible, plain food, supplemented by abundant, nutritious fluids, will but seldom fail to agree with her infant; or, conversely, if the breast milk is scanty or appears to disagree with her infant, either the mother is out of health, anemic, or constipated, she is securing too little fresh air and exercise, she is taking too little fluid food of the right kind, or she is not upon a plain, sensible diet. Surely the busiest practitioner has the time to enquire into these details and to remedy the errors, even if he has no time or facilities for analyses of the breast milk. Were this always done, and especially if proper instructions were given to each mother during her convalescence, far fewer children would be needlessly deprived of the nourishment intended for them by nature.

#### **DISTURBANCES OF THE INFANT DURING LACTATION.**

The principle has been laid down above that the hygiene or the diet of the mother is usually at fault where a healthy mother does not satisfactorily nurse her infant, and that the first duty of the physician in such a case is to correct these errors, which will often alone serve to remove the difficulty. At the same time careful inquiry should be made into the nursing habits, and too frequent or irregular nursings, which directly influence the quality of the milk, should be corrected. Also an analysis of the milk should be obtained where it is possible, and if either fat or proteids vary distinctly from the general averages contained in the table, special measures may be adopted to influence them if relief is not obtained from the changes which have already been inaugurated. The first step would be to determine whether the quantity of the milk is sufficient, and should lead to an inspection of the breasts to determine whether they fill properly during the nursing intervals. Large, fat breasts do not always secrete well, nor small breasts necessarily give an inadequate supply. The mother's sensation of fulness or emptiness of the breasts may be relied upon but moderately. The facility with which the milk may be drawn by the breast pump is often a valuable clue, but the plan of weighing the baby upon a scale which registers ounces, immediately before and after nursing without any change of its garments, and repeating this a few times at different hours of the day, gives the most accurate data. Much may be learned also from the behavior of the infant at the breast and from its stools. If nursing is prolonged thirty minutes or more the milk is probably scanty; or if the infant, after a few minutes at the breast, drops the nipple in disgust and cries, the breast is probably empty.

Restlessness, disturbed or short sleep, associated with frequent stools which consist principally of dark-green mucus with very little fecal matter, in small flakes the size of flattened oats, indicate an inadequate

supply. The quantity of residue and the deep green color of the ~~mucus~~ differentiate this condition from a diarrhea, especially if the ~~feces~~ have the normal orange-yellow color.

### ABNORMALITIES OF BREAST MILK.

*The abnormalities of breast milk may be:* (1) Normal flow with excess of either fat or proteids or of both. (2) Normal or excessive flow with deficiency of fat or proteids or of both. (3) Scanty flow of good quality. (4) Scanty flow of poor quality.

The usual rules given to meet special indications are as follows:

*To increase quantity:* Give mother more nutritious fluids in her diet, especially cornmeal-gruel, and reassure her, if anxious.

*To increase the fat:* Give more meat and possibly prescribe malt extract (not beer).

*To decrease the fat:* Reduce the meat in the diet.

*To increase the proteids:* Give more meats, eggs, and cereals, and lessen the exercise taken.

*To decrease the proteids:* Order exercise by walking, short of fatigue, that the proteids may be used up and not secreted.

In almost every case other matters of health and hygiene will require attention, such as anemia or constipation, as above mentioned. All sources of nervous strain should be removed if possible. Anxiety concerning the ability to nurse must be relieved by cheerful reassurance. If the mother's sleep is broken the infant should be removed at night to another room, out of her hearing. Many mothers, from a sentimental feeling of duty or from lack of assistance, rarely get out into the open air. This reacts upon their health. In the interest of her infant the mother should daily seek out-door exercise and fresh air as a matter of routine. Lactation should not be made a drudgery. If the short nursing intervals leave no time for simple recreation, this should be made possible by one or even two bottle feedings at hours which will give the mother the greatest freedom. A nursing baby thus trained to take one bottle a day is insured against mishaps in case of illness of the mother or departure of the wet-nurse, and the knowledge and apparatus are at hand against such an emergency. Wet-nurses especially are more tractable when they know that the infants can take other food and are not absolutely dependent upon them. Much difficulty is often obviated when an infant has become accustomed to taking food from an artificial nipple. Were a mother's milk absolutely disturbs her baby we should have no hesitation in removing the infant temporarily from the breast and placing it upon a low formula such as it may be reasonably sure to digest, pumping and massaging the breast pending resumption of the nursing. The risk of digestive disturbance or of slight loss of weight, if we begin with a very weak food, is usually less than from continuing the breast milk. Such acute disturbances are, however, comparatively rare.



**DISTURBANCES OF BREAST-FED INFANTS.**

Of the infants presented to us with the story that they are not doing well at the breast the majority fall into two classes: (A) those whose nutrition is good, showing that they have made substantial gains since birth; (B) those whose nutrition is much behind that of the normal breast-fed child of the same age. In Class A the good nutrition of the infant presages prompt improvement under appropriate treatment as indicating a fair supply of breast milk, and the vomiting, poor stools, disturbed sleep, etc., which have caused anxiety, will usually disappear with rigid regulation of nursing intervals and nursing habits and attention to the mother's health and diet. Weaning, too, often proposed in these cases, should not be considered, especially if the infant is gaining in weight. Class B constitutes the more difficult cases, since the poor nutrition indicates probably a poor and insufficient secretion of breast milk. Yet in these cases, unless the mother's health definitely demands weaning, much may often be accomplished in improving the milk so that successful nursing may be carried on in part if not for all of the infant's feedings. Scanty breast milk is not necessarily bad breast milk, and the importance to the infant of the maternal milk is so great that for many reasons it should not be withdrawn if avoidable. Such infants should be under frequent observation and be weighed at intervals of two or three days upon scales which weigh ounces. If such weighings show that the child's weight is stationary, or that it is only losing an occasional ounce, we may safely await the effects of the nourishing fluids and the improved diet and health of the mother. Should, however, the condition of the infant be too serious and its loss of weight be too rapid, then from two to four suitable bottles, beginning with a weak modification of cows' milk, should be given daily, alternating with the breast, and the infant should always be put to both breasts at each nursing in order that the stimulation of these glands by the child may not be lessened. As the mother's milk increases the number of bottles may be lessened and more nursings given. In a fair proportion of such cases they may later be dropped entirely, but more frequently we are satisfied to have the mother nurse the infant in part and supplement her efforts by the requisite number of bottles. In institutions provided with milk laboratories, where usually one mother nurses two babies, the insufficient supply of breast milk is often supplemented by a few ounces of modified milk given immediately after each nursing, the infant's appetite regulating the amount taken. This plan, which works well in institutions, is rather too cumbersome for use in the home, since it involves as much care and preparation as entire artificial feeding; therefore, a few supplementary bottles replacing the same number of nursings is usually adopted. This latter method has also one distinctive advantage—namely, that when bottles and breast are alternated, a longer interval for digestion occurs between the two bottles and there is decreased liability to disturbance. More attention than

formerly is now given to the importance of conserving even a limited amount of breast milk, provided it can be made of reasonable quality, since not only are such children relatively less liable to disease than those who are entirely bottle-fed, but also because, if digestive and bowel disturbances do occur, the child may be temporarily nourished with the more easily digested breast milk. So great is the importance to the infant of maintaining the physiological relation existing between mother and infant until the latter part of the first year, that the physician who counsels early weaning without making all reasonable efforts to enable the mother to continue nursing successfully is assuming a grave responsibility for the life of the child. Bottle-fed children not only suffer more commonly from various forms of malnutrition and digestive disturbances, but have, as a rule, distinctly less resistance to intercurrent disease. Many bottle-fed infants die from maladies which they would have been able to survive had they been nursed. Moreover, during disturbances attendant upon bottle feeding the child often lays the foundation for both digestive and physical difficulties which not only handicap it in later childhood, but may pursue it into adult life.

#### **SPECIAL INFLUENCES WHICH AFFECT THE BREAST MILK.**

**Drugs.**—The more abnormal the secretion of the breast the greater is the liability to the elimination of drugs through this channel. While few definite rules can be laid down, such a possibility should be always kept in mind. Opium, belladonna, and colchicum are especially to be guarded against. When the milk is poor and particularly with young infants, mercury, arsenic, iodides, bromides, lead, antimony and the salicylates at times appear in the milk, especially after prolonged administration. This is not sufficiently constant to be relied upon in attempting to medicate the child through the medium of the mother. Cathartics given the mother at times act upon the infant, and malt beverages, or any considerable ingestion of alcohol, may produce disturbance.

**Menstruation.**—The return of the menstrual flow constitutes, as a rule, no contraindication to a continuance of lactation. In a very small proportion of cases only is the infant disturbed, and in these it is a simple matter to give the infant an easily absorbed substitute and to pump the breasts for a few days. After appearing once, it may be absent for several months. The more discomfort and nervous disturbance the mother experiences the greater the liability to alteration in the secretion, which at times takes the form of low fat and high proteids, although this has not been generally established. The average mother, however, may disregard menstruation and continue nursing during the period.

**Pregnancy.**—The occurrence of pregnancy is almost universally accepted as an indication for taking the child from the breast. The secretion usually suffers both in quantity and quality and the mother's reserves are needed for the nourishment of the fetus. There is, however,

no urgent haste in the matter, if the infant is not losing weight, so that weaning may be gradually and safely accomplished. Other considerations, such as the presence of hot weather and the age and condition of the infant, must influence our decision, but as soon as practicable the mother should be enabled to devote her strength entirely to her new responsibilities.

**Nervous Influences.**—Every observant dairyman will inform us that the milk of the cow is easily affected by nervous influences. This is equally true of the nursing mother, whose life should, so far as possible, be a passive one, free from undue excitement or mental worries and anxieties. To this end she should be willing to surrender, for the sake of her infant, all those social responsibilities and dissipations which are the source of fatigue and nervous wear and tear. Instances are recorded where intense or sudden emotion, fear, grief, mental shocks, and mental and physical passion have induced a toxic condition of the milk, with grave disturbances in the infant. These are probably the result of some change in the proteids as yet undetermined, since the symptoms—vomiting, diarrhea, temperature, stupor, and even convulsions—are similar to those from other toxins of proteid origin. Reasonable diversion should, on the other hand, not be denied the mother, and out-of-door exercise without fatigue is of the highest importance.

**Minor Acute Illness.**—The flow of milk may be decreased by temperature, but the lesser temporary ailments, even if rather acute, do not seriously affect the milk, and at most call for temporary removal from the breast.

**Severe Acute Illness.** Typhoid and other prolonged fevers call for cessation of nursing and, indeed, often dry up the breasts. Severe general sepsis in the mother is a menace to the infant, owing to the presence of micro-organisms in the maternal circulation and the milk; but slight, local puerperal infection of short duration constitutes no bar. In case of abscess of one breast the infant should not be allowed to nurse that breast when suppuration is probable, nor when pus is demonstrable to the eye or to the microscope. Nursing may be resumed as soon after incision as the cavity shows healthy granulations which close the milk-ducts. The infant may continue at the sound breast, and the affected breast, if possible, be pumped to maintain the secretion. Early incision limits tissue destruction, shortens the inflammatory process, and enhances the possibilities of a useful breast.

**Chronic Illness.** Certain maladies of the parent render nursing inadvisable in the interest of the mother or the child, or both. Such are, dementia, epilepsy, tuberculosis, and marked albuminuria. In the two latter, nursing favors the progress of the disease, while the milk secreted will probably be of inferior quality. In tuberculosis the opportunities for infection of the infant are greatly enhanced by close association with the person of the mother. Danger of direct infection through the milk increases with the advance of the disease and the consequent liability of the occurrence of tuberculous foci in the mammae. A syphilitic infant, on the contrary, is more liable to survive if nursed by its mother, who,

according to Colles' law, is immune to the disease. A wet-nurse, however, should always be free from any suspicion of such taint, and may readily become infected by nursing a syphilitic child.

### WEANING.

No definite rule as to the proper time for weaning a child can be laid down, but the general statement may be made that with the average healthy mother in America it should be undertaken at about the end of the first year. Few mothers can nurse their children to advantage longer than this. Many, on the other hand, cannot maintain a satisfactory supply so long. In the latter case I have already recommended measures for supplementing the breast with bottle feedings, and in such instances weaning is accomplished easily, since the gradual decrease of breast milk leads to the giving of more and more bottle feedings. If we can choose the time of weaning, this may then be from the ninth to the twelfth month, but preferably not in the hot months of the year, for in midsummer it is better to defer the completion of weaning a few weeks until the cooler days of early autumn, giving, perhaps, two bottles daily to eke out the breast milk. At such a time even a stationary weight without loss should not deter us from waiting two or three weeks, since, if digestive disturbance can be avoided, children rapidly make up their weight on the new food in the fall months, and in the event of illness we have the breast milk to fall back upon temporarily. In the late spring it may be best to get the child accustomed to taking one or two bottles a day before the hot weather supervenes, if the breast milk will probably have to be withdrawn before the fall.

The keynote of safe weaning is that the process be gradual, and also to keep in mind the fact that cows' milk is a different fluid, which the child must be trained to digest. To this end we must begin with a low formula for a child of nine months or one year old, not exceeding 1 per cent. of proteids, and for younger children 0.50 to 0.75 per cent. proteids, increasing these promptly as the stools show proper digestion. (See page 141.) Such formula should at first be given like supplementary feedings, once or twice a day, and, when the formula has been raised to one fitted for the age, others are added, one after the other, until the breast is largely supplanted by the bottle. As less demands are made upon the breast its secretion usually disappears without trouble. With older children the time of weaning is the time for the introduction of cereals into the diet, and these are best incorporated into the diet in the form of barley, oatmeal, or grannum. Some children can undoubtedly soon be brought to take plain milk, but in many cases this is not as easily digested as that diluted one-third to one-fifth with a cereal gruel, and there is an advantage to be gained from the use of cereals at this time and in this form. Since milk is to be for many months the basis of the diet, and since children without question will drink more from a

bottle than from a cup or glass, the bottle is preferable if the child can be induced to take it. Here, again, the child should not be limited by the size of the usual 8-ounce (250 c.c.) feeding bottle, but 9 to 12 ounces (280 c.c. to 375 c.c.) should be given from a larger bottle, according to the age. In suitable cases broths or beef-juice may be given once daily. In this way the weaned child gradually takes up the diet suitable for the second year.

## CHAPTER VII.

### COWS' MILK.

THE practitioner who to-day wishes to become expert in modern infant feeding must necessarily have a good general knowledge not only of the chemistry and bacteriology of cows' milk, but oftentimes be able to advise intelligently concerning its production, since the solution of many of the problems to be met with is directly based upon these factors. The dairy interests of the world have assumed such importance among its productive resources that elaborate studies of these questions have been made possible under governmental and university auspices in this and other countries.

**Composition.**—Milk is composed principally of water, fat, sugar, proteid or albuminoid bodies, and mineral matter. The fat is suspended as a fine emulsion with the other elements, and is a mixture of several fatty compounds. By far the largest proportion (92 per cent.) is fixed and non-volatile, and consists of glycerides of oleic, stearic, and palmitic acids. A smaller volatile group, of which the most important is butyric acid, is constant, and constitutes but 8 per cent. Still others may be derived from special foods consumed, and the flavors given to milk by cabbage, onions, and turnips as well as the more desirable ones imparted by clover and grasses are due to such volatile fats.

**Proteids.**—These, sometimes called albuminoids, are casein and certain soluble albuminous bodies. Casein is thrown down by the action of rennet and also by weak solutions of acids, making what is loosely known as the curd. The soluble proteids consist of several bodies which are not influenced by either rennet or weak acids, but are to some extent coagulated by heat, forming the familiar skin on boiled milk. They are contained in the whey which separates from the paracasein clot formed by rennet, and in the fluid which is pressed out from the curds of fully soured milk known as cottage cheese.

In older books, and even in some of the modern ones, the terms lactalbumin, lactoglobulin, lactoprotein, albumin, caseoses, albumoses, and peptones are variously applied to these or they are referred to as albumins. In these pages they will be spoken of collectively as the soluble proteids of milk. This term will also be applied to the proteids remaining in solution in whey, in the clear fluid which separates from the coagulum of sour milk, and after the precipitation of the casein in digestion.

The analytical methods devised thus far for the proteids of milk give only approximately accurate quantitative results. In addition to the proteid bodies already mentioned there is another analogous to mucin.



This is usually called Storch's mucoid proteid, and is profoundly affected by lime-water and other strong alkalies, which cause it to swell and become viscid, thus visibly thickening the milk. It is not affected by non-alkaline antacids, such as chemically pure bicarbonate of soda.

**Sugar.**—The lactose, or milk-sugar, is held in solution. It has the same chemical formula as cane-sugar, but is distinctly less sweet to the taste.

**Ash.**—The mineral matters of the milk are designated in analyses as the ash. They are chiefly in solution and consist in large part of phosphates of calcium and potassium, chlorides of potassium and sodium, and small quantities of phosphates of iron and manganese.

**Water.**—Water constitutes a large proportion of milk, being from 84 per cent. to 88 per cent. of the whole.

Milk also contains very small quantities of other bodies, of less importance to the practitioner, such as citric acid, lecithin, and certain enzymes having the property of slightly digesting milk. The foregoing constituents of cows' milk vary widely in different specimens. The variations are dependent upon the country, the breed of cattle, the period of lactation, the diet, the time of day, and the intervals between milkings.

#### VARIATIONS IN MILK.

A brief discussion of the variations in cows' milk will be instructive. If the interval between the daily milkings differ in length, the longer period will give a lower percentage of fat in the milk, since some of the fat is reabsorbed during its retention in the udder. This is likewise true of the human breast and furnishes a cogent reason for adhering to regular intervals in nursing. Short intervals give a milk too rich in fat. The composition of different portions of the cow's milk when drawn from the udder is not the same as that of the whole milking in the pail. The first milk drawn (foremilk) contains the least fat, often less than 2 per cent.; while that drawn last, called strippings, may reach as high a percentage as 10 per cent. The other solids do not vary materially during milking. Aside from the above factors, the variations in the composition of the whole milk of individual cows are principally due to nervous influences. These are not only such as fright and worry, but even sudden changes in the food of highly bred cows probably act in this way through the nervous system (Babcock). The less highly bred and less nervous animals are not so readily affected by any change and the composition of their milk is more uniform. Gradual changes in the food do not materially affect the milk of a healthy cow. Proper feeding may increase the quantity of milk, but the almost unanimous opinion of dairy experts is that it is beyond our power to alter the character or composition of a healthy cow's milk from that which is normal for the individual cow by any means except nervous influences. The good results obtained in changing the composition of human breast milk are due to the restoration of healthful conditions and the removal of pernicious nervous

influences which allow the re-establishment of a secretion of milk which is normal for that mother.

Possible individual variations in cows' milk are equalized by mixing the milk of a herd. The composition of such mixed milk remains fairly constant at the same season of the year. That of different herds varies with the breed of the cows composing the herd, the fat being highest in the highly bred Jersey and Guernsey stock. In general the milk of a well-cared-for herd of good grade cows is to be preferred for infant feeding to that of fancy stock with delicate, nervous organizations and a high percentage of fat in the milk.

It is therefore to be understood at the outset that cows' milk as produced or sold has no fixed composition, the local laws governing its sale being adjusted so as to do no injustice to the farmer producing only a fair, unadulterated milk.

From a large series of analyses made by Van Slyke, and representing individual milks whose fat percentages ranged from 3.05 per cent. to 5.25 per cent., the following have been selected to show the composition of poor, medium, and rich milks:

ACTUAL ANALYSES OF POOR, MEDIUM, AND RICH MILKS.

	Poor milk.		Medium milk.		Rich milk.
Fat . . . . .	3.66		4.05		5.00
Total proteids . . . . .	2.64	{ casein, 1.98. soluble proteids, 0.66.	3.45	{ 2.77 0.68	3.39 { 3.14 0.85
Sugar and ash . . . . .	5.91		5.59		5.64
Water . . . . .	88.40		86.91		85.37

These actual analyses are much more instructive to the practitioner than the usual tables which contain averages made by combining the results of many hundreds or thousands of analyses of milks of all qualities. The above table shows the relative percentages of the total proteids and their component parts—casein and soluble proteids—in specimens of milk which contained 3 per cent., 4 per cent., and 5 per cent. of fat. The sugar and ash are calculated together in these tables, but, since the ash of milk is known to be fairly constant at 0.70 to 0.75, the percentage of milk-sugar (lactose) in milk can be set down at about 5 per cent. In general it may be said that the sugar and ash are fairly constant and the fat and proteids variable factors. As a rule, the amount of proteid rises and falls with the amount of fat, but increase of fat beyond 4.5 per cent. does not involve a proportionate increase of proteids.

The practitioner will, if possible, usually choose, in preference to all others, a good milk containing about 4 per cent. fat, which may be determined with the Babcock milk-testing machine. A table constructed upon this basis would be as follows:

AVERAGES OF GOOD MILK CONTAINING 4 PER CENT. FAT.

Fat . . . . .	4.00
Total proteids . . . . .	3.50
Sugar . . . . .	5.00
Ash . . . . .	0.70
Water . . . . .	86.80

**BACTERIAL CONTAMINATION OF MILK.**

Milk is one of the favorable culture media at certain temperatures for the growth of bacteria. While milk when secreted by a healthy cow is germ free, bacteria are always present in the larger ducts, to which they penetrate from without. The number in the milk may be largely reduced by rejecting the first few streams of milk, but the chief contamination occurs after it leaves the udder. The universality of bacterial occurrence and the startling rapidity of bacterial growth and multiplication are exceedingly difficult matters to grasp without actual personal experience in the laboratory, and their importance is but imperfectly realized by the lay mind, to which the subject is necessarily a vague and confused one. It is only when these questions come to affect the profit or loss of the dairy business that an intelligent and practical interest can be aroused. Bacteria are now definitely known to be vegetable organisms. In those forms which are spore bearing life is much less easily destroyed by extremes of temperature. Rapid development is checked by low temperatures. Freezing reduces the number of the bacteria, but does not kill all, especially the spore-bearing forms, and upon thawing they again become active. Growth and multiplication proceed most rapidly in warm media, but extreme heat kills at a temperature varying with the resistance of the individual bacterium. It may be said of most of the usual bacteria in milk that growth is checked at a temperature below  $40^{\circ}$  to  $50^{\circ}$  F., is most favored by a temperature of  $80^{\circ}$  to  $100^{\circ}$  F., again decreases at  $105^{\circ}$  to  $110^{\circ}$  F., and with the exception of the spore-bearing forms have a thermal death point of  $130^{\circ}$  to  $140^{\circ}$  F. in liquids. Spores may require the action of superheated steam to kill them. Not only does milk inevitably contain some bacteria which have gained access to the ducts despite the rejection of the first streams, but bacteria which are practically omnipresent may be added in every phase of the milking and subsequent handling of the milk as carried out under ordinary conditions.

No particle of matter, however small, seems to be free from germ life. Dirt, fecal matter, hair, the floating dust of the barn, the surface of the milker's hands and clothes, receptacles for milk, even if apparently clean, all furnish their quota. Not only does the active exercise of milking dislodge dust, dirt, and dandruff from the cow, which may fall into the pail, while the hands and clothing of the milker contribute their share; but particles of dust in the air, which are much increased by the manipulation of dry fodder and bedding, constantly settle into the milk-pail. The dust in the air is considered one of the minor factors only, though Harrison's experiments with culture plates showed that in one minute, during the process of bedding down, from 12,000 to 43,000 bacteria settled on a surface equal to that of a 12-inch milk-pail, while an hour before bedding a similar series of cultures showed from 483 to 2370.

Housing the cow in a barn away from all hay and fodder, keeping the cow's body and stable free from filth and dust, cleansing and dampening



the udder and teats before milking, washing the hands before milking, rejecting a small part of the foremilk, and sterilization of the milk receptacles and utensils as now practised in the best dairies, reduce vastly the germ content of the milk, and Backhaus considers that these measures will easily give only  $\frac{1}{2000}$  of the number of germs in milk produced in the usual way. The subsequent care of the milk calls for straining and rapid cooling, with the same precautions against contamination. Aeration, formerly considered necessary to remove animal odors from the milk, is now thought to be sufficiently accomplished during milking, thus avoiding an additional manipulation with its attendant risks. Straining removes the coarser particles of dirt, but not that which has been dissolved in the milk with its bacteria. Rapid cooling limits the multiplication of germ life, which goes forward with startling rapidity until a sufficiently low temperature has been reached. This temperature must be maintained continuously during all the phases of transportation and distribution, since growth is at once accelerated if the temperature is allowed to rise. Bottling at the farm in sealed, sterile containers is the only guarantee against contamination *en route*.

**Certified Milk.**—The education of the medical profession and the laity with reference to the advantages of clean milk of low bacterial content has led to the establishment in various places of systems by which local health boards or duly organized groups of physicians have undertaken to place the seal of their approval upon certain dairies whose milk comes up to a required standard of cleanliness.

Such milk bears a label, and may be known by some name such as "certified milk." In other localities, certain large, private, model dairies have created their own reputation for the purity of their milk, which is borne out by repeated bacteriological tests.

Since the physician may at any time be called upon to pass judgment upon the conditions under which milk is produced, or to give instructions which shall safeguard milk which is to be used in infant feeding, certain abstracts from the directions issued by the Milk Commission of the Medical Society of the County of New York, which certifies milk which comes up to its requirements, are here given.

**Rules for Production of Certified Milk.**—The most practicable standard for the estimation of cleanliness in the handling and care of milk is its relative freedom from bacteria.

"The Commission has fixed upon a maximum of 30,000 germs of all kinds per cubic centimetre of milk, which must not be exceeded to obtain the endorsement of the Commission. This standard must be attained solely by measures directed toward scrupulous cleanliness, proper cooling, and prompt delivery.

"The milk certified by the Commission must contain not less than 4 percent. of butter-fat on the average, and have all other characteristics of pure, wholesome milk.

"Milk must not be sold as certified more than twenty-four hours after its arrival in New York City.

"The required conditions are as follows:

"1. **THE BARNYARD.**—The barnyard should be free from manure and well drained, so that it may not harbor stagnant water. The manure which collects each day should not be piled close to the barn, but should be taken several hundred feet away. If these rules are observed, not only will the barnyard be free from objectionable smell, which is an injury to the milk, but the number of flies in summer will be considerably diminished.

"These flies are an element of danger, for they are fond of both filth and milk, and are liable to get into the milk after having soiled their bodies and legs in recently visited filth, thus carrying it into the milk.

"Flies also irritate cows, and by making them nervous reduce the amount of their milk.

"2. **THE STABLE.**—In the stable the principles of cleanliness must be strictly observed. The room in which the cows are milked should have no storage loft above it; where this is not feasible the floor of the loft should be tight, to prevent the sifting of dust into the stable beneath.

"The stables should be well ventilated, lighted, and drained, and should have tight floors, preferably of cement, never of dirt.

"They should be whitewashed inside at least twice a year, unless the walls are painted or of smooth cement finish, which can be washed frequently.

"The air should always be fresh and without bad odor. A sufficient number of lanterns should be provided to enable the necessary work to be properly done during the dark hours. The manure should be removed twice daily, except when the cows are outside in the fields the entire time between the morning and afternoon milkings. The manure gutter must be kept in a sanitary condition. All sweeping must be finished before the grooming of the cows begins, so that the air may be free from dust at the time of milking.

"There should be an adequate supply of water, warm and cold, and the necessary wash-basins, soap, and towels.

"3. **WATER SUPPLY.**—The whole premises used for dairy purposes, as well as the barn, must have a supply of water absolutely free from any danger of pollution with animal matter, and sufficiently abundant for all purposes and easy of access.

"4. **THE COWS.**—No cows will be allowed in the herd furnishing certified milk except those which have successfully passed a tuberculin test. All must be tested at least once a year by a veterinarian approved by the Milk Commission. Any animal suspected of being in bad health must be promptly removed from the herd and her milk rejected. Do not allow the cows to be excited by hard driving, abuse, loud talking, or any unnecessary disturbance.

"*Feed.*—Do not allow any strongly flavored food, like garlic, to be eaten by the cows.

"When ensilage is fed it must be given in only one feeding daily, and that after the morning milking, and the full ration shall consist of not more than twenty pounds daily for the average-sized cow. When fed

in the fall small amounts must be given and the increase to the full ration must be gradual.

"Corn stalks must not be fed until after the corn has blossomed, and the first feedings must be in small amounts and the increase must be gradual.

"If fed otherwise, ensilage and corn-stalks are liable to cause the milk to affect children seriously.

"*Cleaning*.—Groom the entire body of the cow daily. Before each milking wash the udder with a cloth used only for the udders, and wipe it with a clean, dry towel. Never leave the udder wet, and be sure that the water and towel used are clean. The tail should be kept clean by frequent washing. If the hair on the flanks, tail, and udder is clipped close and the brush on the tail is cut short it will be much easier to keep the cow clean.

"The cows must be kept standing after the cleaning until the milking is finished. This may be done by a chain or a rope under the neck.

"5. THE MILKERS. The milker must be personally clean. He should neither have nor come in contact with any contagious disease while employed in handling the milk. In case of any illness in the person or family of any employé in the dairy, such employé must absent himself from the dairy until a physician certifies that it is safe for him to return.

"In order that the Milk Commission may be informed as to the health of the employés at the certified farms, the Commission has had postal cards printed, to be supplied to the farms, and to be filled out and returned each week, by the owner, manager, or physician of the farm, certifying that none are handling the milk who are in contact with any contagious disease.

"Before milking the hands should be washed in warm water with soap and nail brush and well dried with a clean towel. On no account should the hands be wet during milking.

"The milkers should have light-colored, washable suits, including caps, and not less than two clean suits weekly. The garments should be kept in a clean place, protected from dust, when not in use.

"Iron milking stools are recommended, and they should be kept clean.

"Milkers should do their work quietly and at the same hour morning and evening. Jerking the teat increases materially the bacterial contamination of the milk and should be forbidden.

"6. HELPERS OTHER THAN MILKERS.—All persons engaged in the stable and dairy should be reliable and intelligent. Children under twelve should not be allowed in the stable or dairy during milking, since in their ignorance they may do harm, and from their liability to contagious diseases they are more apt than older persons to transmit them through the milk.

"7. SMALL ANIMALS.—Cats and dogs must be excluded from the stables during the time of milking.

"8. THE MILK.—All milk from cows sixty days before and ten days after calving must be rejected.



"The first few streams from each teat should be discarded, in order to free the milk-ducts from the milk that has remained in them for some time and in which the bacteria are sure to have multiplied greatly. If any part of the milk is bloody or stringy or unnatural in appearance, the whole quantity yielded by that animal must be rejected. If any accident occurs in which a pail becomes dirty, or the milk in a pail becomes dirty, do not try to remove the dirt by straining, but put aside the pail, and do not use the milk for bottling, and use a clean pail.

"Remove the milk of each cow from the stable immediately after it is obtained to a clean room and strain through a sterilized strainer of cheese-cloth and absorbent cotton.

"The rapid cooling is a matter of great importance. The milk should be cooled to 45° F. within an hour and not allowed to rise above that as long as it is in the hands of producer or dealer. In order to assist in the rapid cooling, the bottles should be cold before the milk is put into them.

"Aeration of milk beyond that obtained in milking is unnecessary.

"9. **UTENSILS.**—All utensils should be as simple in construction as possible, and so made that they may be thoroughly sterilized before each using.

"Coolers, if used, should be sterilized in a closed sterilizer, unless a very high temperature can be obtained by the steam sent through them.

"Bottling machines should be made entirely of metal with no rubber about them, and should be sterilized in the closed sterilizer before each milking or bottling.

"If cans are used, all should have smoothly soldered joints, with no places to collect the dirt.

"Pails should have openings not exceeding eight inches in diameter, and may be either straight pails or the usual shape with the top protected by a hood.

"Bottles should be of the kind known as 'common-sense,' and capped with a sterilized paraffined paper disk, and the caps authorized by the Commission.

"All dairy utensils, including the bottles, must be thoroughly cleansed and sterilized. This can be done by first thoroughly rinsing in warm water, then washing with a brush and soap or other alkaline cleansing material and hot water, and thoroughly rinsing. After this cleansing they should be sterilized by boiling, or in a closed sterilizer with steam, and then kept inverted in a place free from dust.

"10. **THE DAIRY.**—The room or rooms where the utensils are washed and sterilized and the milk bottled should be at a distance from the house, so as to lessen the danger of transmitting through the milk any disease which may occur in the house.

"The bottling-room, where the milk is exposed, should be so situated that the doors may be entirely closed during the bottling and not opened to admit the milk nor to take out the filled bottles.

"The empty cases should not be allowed to enter the bottling room nor should the washing of any utensils be allowed in the room.

"The workers in the dairy should wear white washable suits, including cap, when handling the milk.

"Bottles must be capped as soon as possible, after filling, with the sterilized disks.

"11. EXAMINATION OF THE MILK AND DAIRY INSPECTION.—In order that the dealer and the Commission may be kept informed of the character of the milk, specimens taken at random will be examined weekly by experts for the Commission, at the Laboratory of the Department of Health, the use of the laboratories having been given for that purpose.

"The Commission reserves to itself the right to make inspections of certified farms at any time and to take specimens of the milk for examination, and to impose fines for repeated or deliberate violations of the requirements of the Commission.

"The Commission also reserves the right to change its standards in any reasonable manner upon due notice being given to the dealers.

"The expenses of making the regular milk reports and the inspections are borne by the dealers."

Experience has shown that the periodical examinations, which include estimations of the number of bacteria per cubic centimetre of milk, are necessary not only for control, but also prove a great incentive at the dairy to improve the technique of production so as to lower the bacterial count. Under the best conditions of production, milk still contains several thousands of bacteria per cubic centimetre, and while this is a vast improvement over conditions which allow of the sale of milk in cities which contain at some seasons of the year anywhere from 1,000,000 to 50,000,000 bacteria per cubic centimetre, sight should never be lost of the fact that bacteria and their action must always be reckoned with in the consideration of milk as a food. Many of these bacteria are innocuous, others have a fermentative or putrefactive action, while still others are pathogenic and capable of producing disease. The degree of danger which arises from the use as food of the milk of a tuberculous cow is still a mooted question; but since undoubted instances of the transmission of tuberculosis by this means have been recorded, ordinary common-sense dictates that tuberculosis should be eliminated from the herds and the milk of such cattle rejected. With tuberculosis of the udder the tubercle bacilli may be demonstrated in the milk. The use of the tuberculin test to eliminate diseased animals from the herd should be encouraged.

**Epidemics.**—Epidemics of aphthous stomatitis have been traced to the drinking of milk from cows suffering from foot-and-mouth disease. Tetanus, anthrax, and hydrophobia are other diseases of cattle to which man is liable, but the only rule for safety is to reject the milk of any cow which shows evidence of illness. Epidemics of typhoid fever, scarlet fever, diphtheria, and cholera from contamination of the milk by persons employed in its handling, or from water used in diluting it or for washing the utensils, are fully authenticated.

**Bacteria of Putrefaction.**—There remain to be considered the bacteria which gain access to the milk in the usual way during its production.

These, which are of many different kinds, are roughly divided into putrefactive and fermentative groups. The former—the putrefactive—act upon the proteids, and certain of them may occasionally form toxins in the milk before it is consumed, which, when taken into the system, cause severe and even fatal poison. Such a substance isolated by Vaughan has been called tyrotoxicon. Other putrefactive bacteria may find special conditions for their development after reaching the digestive tract, especially if digestion is disturbed. This is doubtless the source of some of the more intense cases of the so-called summer diarrhea of young children, and since the proteids of milk furnish a suitable material for the development of such bacteria and their elaborated toxins, the rule is now absolute to stop milk in any form and to evacuate its residue thoroughly from the bowel upon the appearance of diarrhea.

**Bacteria of Fermentation.**—Of the fermentative forms the so-called lactic-acid-producing bacteria are the most important. Although when the milk is drawn these may be in the minority, ordinary conditions to which milk is subjected are so much more favorable to their growth that they crowd the others into the background, and are soon more than 90 per cent. of the bacteria in the milk. Their rapid growth is favored by the presence of the milk-sugar, which they transform into lactic acid. This lactic acid increases with the multiplication of the bacteria until it causes coagulation of the casein, and the most common change, therefore, in milk is that of souring. Such a change renders it unfit for infant feeding, although it is often easily digested by adults. Souring of milk formerly ascribed to thunder-showers can only be explained by the fact that the atmospheric conditions and temperature before the storm favor this fermentation.

Not only do acids form definite chemical products with calcium paracasein (calcium casein clotted by rennet), but acids, including lactic acid, may act upon calcium casein, directly forming both free casein and a compound of casein and acid. Lactic acid in small amount does not at first precipitate the casein, but as it increases, and especially if the milk is warmed, there appear in the milk fine flocculi which are chiefly free casein. When the lactic acid reaches about 0.6 to 0.7 per cent. (total acidity 0.8 to 0.9 per cent.) the milk forms a semisolid mass or clabber, which is chiefly lactate of casein, and the growth of the lactic bacteria soon ceases. Before the acid has increased to the point of precipitating the casein, rennet may still act upon the calcium casein, forming calcium paracasein, which with the acid present is changed into free paracasein and lactate of paracasein. These, like all forms of paracasein produced by the action of acids, are tough, slowly contracting masses. When, however, lactic acid has precipitated the calcium casein in soft masses of free casein and lactate of casein, rennet can no longer act upon these and the denser paracasein compounds cannot be formed. The products of calcium casein and acids, being softer than those of calcium paracasein and acids, are then usually more digestible, but products of either with lactic acid do not apparently differ materially in their relative digestibility from similar combinations formed by hydro-



chloric acid. Fully soured milk, clabber, and buttermilk are readily digestible for the adult because tough paracasein products cannot be formed from them, since rennet does not affect them. Both kumyss and zoolak, which are the products of types of fermentation with the production of lactic acid, contain soft flocculi precipitated by the acid, and probably owe their digestibility largely to the same principle.

The presence of milk-sugar favors the production of lactic acid, and the latter also holds the activity of the putrefactive groups of bacteria in check.

Heating milk to a sufficiently high temperature to destroy the lactic-acid-forming bacteria does not completely destroy other forms in the milk which contain spores. Such milk undergoes different changes through the action of these unkilld spore-bearing forms, which would have been prevented from developing by the presence of the lactic acid forms. These produce either a curdling of the milk and subsequent digestion of the proteids or digestion without curdling, conditions which are brought about by the action of unorganized ferments or enzymes to which the bacteria give rise. It is for this reason that such heated milk, although it will not sour and therefore can be used longer, will often develop a very foul odor and become poisonous. Milk which has been heated, as well as that which has not been heated, should therefore be kept cool if it is to be used as an infant's food, and should not be subjected to warmth for any length of time.

### MILK PRESERVATION.

The preservation of milk which is to be used as the food of infants is of the highest importance. Since the changes which milk undergoes are in direct proportion to the number of bacteria which it contains, it does not require further argument to demonstrate that a clean milk which has from the outset contained the lowest possible number of germs is vastly preferable to a milk in which germs already present in large numbers have been killed or held in check by artificial methods. Not only are the constituents of the milk altered by the presence of bacteria which are nourished by it and produce in their growth and action by-products which may be both foreign and hurtful, but in the case at least of spore-bearing forms they cannot be destroyed without seriously changing the nutritive and digestible properties of the milk.

The necessity of securing a clean milk being admitted, it is still necessary to consider methods of preservation, since milk from the most unimpeachable sources still contains a rather formidable number of germs.

**Preservatives.**—The question of preservatives can be dismissed with a few words of unqualified condemnation. Milk designed for the feeding of children should not be subjected to any form of chemical adulteration. Alkaline antacids, like sodium bicarbonate, may neutralize acid already formed in the milk, but do not inhibit the growth of the acid-forming

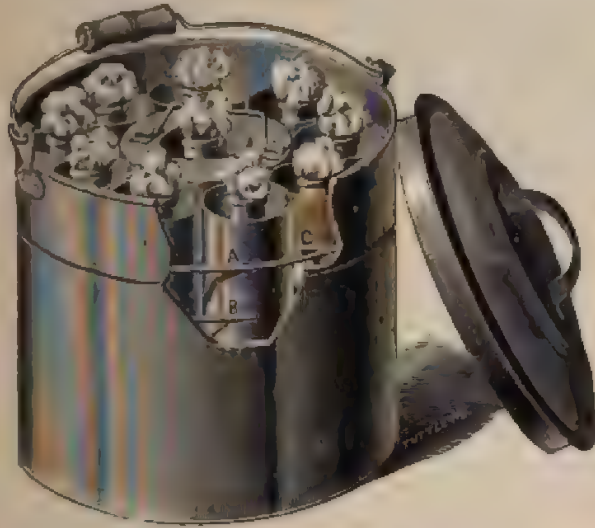
bacteria, but instead favor it, since 1 per cent. lactic acid checks growth. Salicylic acid, boric acid, and formaldehyde, which form the bases of most commercial preservatives, even if they be not added in amounts which are definitely hurtful (and this is still a mooted question), have no place among the requirements of the child. In many foreign countries where the passage of pure food laws cannot be retarded by the cupidity of the dealers the use of such preservatives is absolutely prohibited.

**Accepted Measures of Preservation.**—The trend of the best medical opinion at the present day is undoubtedly toward the use of fresh, clean, unheated milk when conditions are such as to render this safe. Such conditions, however, only exist during the cool months of the year with milk from a healthy inspected herd, handled at every stage with the utmost attention to cleanliness, kept constantly at a temperature below 40° to 50° F., and used at or near its place of production while it is still fresh. Naturally, this combination can only obtain in the country and towns where milk is supplied from the immediate environment. In cities milk is almost invariably, in part at least, twenty-four hours old when it reaches the consumer, and, indeed, some of it often thirty-six or forty-eight hours old. It must then serve for twenty-four hours longer or until the next day's supply arrives. Some measure must, therefore, be adopted to check the already abundant growth of bacteria and to preserve the milk from souring. Three methods are in general use for this purpose—pasteurization, sterilization, and boiling. These terms are much more indefinite than is generally supposed, since temperatures from 60° to 75° C. (140° to 167° F.) are recommended for pasteurization for various periods of time. Sterilization is the term applied to heating to 100° C. (212° F.) or generally to a higher point for different lengths of time. Boiling is often considered to begin when the milk rises in the container at 75° to 85° C. (167° to 185° F.), but it actually boils at about 101° C., with the appearance of large bubbles. Some of the differences of opinion concerning the effects of these methods upon the milk are doubtless thus explained.

**Pasteurization.**—Pasteurization may be carried out in various ways. Theoretically the best way would be to have the milk pasteurized at the dairy in the bottles in which it is delivered to the customer before the germs have had an opportunity to multiply. While this method would be preferable for children old enough to take whole milk it is inapplicable in the majority of cases where the milk is to be modified at the home, since the necessary handling and exposure during modification would render a second heating advisable. Another and even more grave difficulty consists in the previously mentioned fact that pasteurization kills the lactic-acid-producing bacteria, which are inimicable to other forms, and clears the way for the free development of the putrefactive germs; so that such milk, unless handled with the greatest care and kept continuously cool, may in time become more dangerous than if it had not been heated at all. Pasteurization was at first popularized in this country by the efforts of Dr. R. G. Freeman, who devised a practical and

not expensive apparatus for home use known as the Freeman pasteurizer (Figs. 27 and 28), in which the number of nursing bottles required for

FIG. 27



Freeman pasteurizer.

FIG. 28



Freeman pasteurizer.



use in twenty-four hours are, with their contents, raised to a temperature of  $68^{\circ}\text{C}$ . ( $155^{\circ}\text{F}$ .) and maintained at that point for thirty minutes. A cheaper apparatus may be constructed from a large tin pail holding a rack for the nursing bottles, the cover of which is perforated by a hole to admit a chemical thermometer. The bottles are then immersed in water up to their necks and the whole heated until the thermometer registers  $75^{\circ}\text{C}$ . ( $167^{\circ}\text{F}$ .), when it is moved back on the stove and allowed to stand twenty minutes. Again, the whole supply of milk prepared for the day's feedings may be placed in a large, glass fruit-jar closed by a cork, through which passes a chemical thermometer, and the jar surrounded by water and heated to  $75^{\circ}\text{C}$ . ( $167^{\circ}\text{F}$ .), and this temperature maintained twenty minutes, when the cork is replaced by a cap or a plug of sterilized cotton. With the use of any of these methods the receptacles containing the milk should be removed at the end of the period of pasteurization, and cooled as rapidly as possible in running water and then placed on ice. Placing the receptacle directly on ice, without preliminary cooling, wastes ice, and there is a longer period during which the milk remains warm, thus favoring the development of the unkilld spores.

**Sterilization.**—Absolute sterilization of milk can only be secured by heating to points considerably above  $100^{\circ}\text{C}$ . ( $212^{\circ}\text{F}$ .) for one hour on each of three successive days. This method kills the bacteria which germinate in the intervals from the latent spores which have remained unaffected. Sterilization as practised in the home is never complete, for while it kills the living germs it does not kill the spores. However, sterilization at  $100^{\circ}\text{C}$ . ( $212^{\circ}\text{F}$ .) for ten to thirty minutes is practically sufficient when milk is to be used within the following forty-eight hours. This may be carried out by boiling the whole supply in a saucepan or double boiler; or the separate feedings for the day, contained in nursing bottles stoppered with sterilized non-absorbent cotton and held upright in a rack, may be subjected to the action of steam in an Arnold sterilizer, or simply boiled by placing the rack in any covered receptacle containing water. An intermediate process suitable for employment among classes where but little time for the care of the milk can be exacted, and one which embraces some of the advantages claimed for both pasteurization and sterilization, consists in bringing the milk in a saucepan or double boiler just up to the point where boiling commences, removing it from the hot fire, standing it for twenty minutes in a warm place, cooling it rapidly in water, and placing it on ice in a clean, stoppered jar or bottle.

The relative merits and demerits of pasteurization and sterilization, together with the indications for their employment, may be summed up as follows:

**Pasteurization and Sterilization Compared.**—Pasteurization does not alter the taste of the milk, nor change the chemical constituents, nor directly affect materially the digestibility; while it kills the bacteria of tuberculosis, typhoid, diphtheria, cholera, and the pathogenic forms of bacteria, such as the staphylococcus, the streptococcus, and bacillus coli

communis. It also destroys most other forms which are to be found in milk, but does not affect the spore-bearing peptonizing and butyric-acid-forming groups. If the milk is subsequently kept properly cool, it is sufficient to preserve the milk two or three days, or more than ample time for ordinary use in infant feeding. In an indirect way pasteurization may definitely influence the digestibility of the casein of cows' milk. The quantity of tough products of paracasein and acid formed in the stomach is proportionate to the total amount of acids present. Pasteurization by destroying the lactic germs prevents the formation of lactic acid, so readily produced in milk, especially during the summer months. This allows the normal acid of the stomach to form its own amount of paracasein products, which will more probably be in proportion to the digestive powers and the amount of pepsin secreted. Pasteurization may then at least be said to prevent milk from becoming more indigestible. Furthermore, the action of rennet is slower and more imperfect upon pasteurized milk.

Some of the changes which are said to be produced in milk by the higher temperatures included in the term sterilization are decomposition of lecithin and nuclein, reduction of the organic forms of phosphorus, change in form of part of the lactose, greater coalescence of the fat globules, coagulation of the albumin of the soluble proteids, which progresses steadily above  $75^{\circ}\text{C}$ . ( $167^{\circ}\text{F}$ .) and a more imperfect action upon the action of rennet, pepsin, and pancreatin. There is also an alteration in the taste. It would, therefore, seem that certain vital principles are altered or destroyed, and the claim is made, with reasonable probability, that its exclusive use favors the development of anemia, rickets, scurvy, and constipation. Prolonged heating at high temperatures should therefore be discouraged, except where there are good grounds for its use. As in all matters pertaining to infant feeding, judgment and careful consideration of the special circumstances should enter into our choice. Neither method should be made a fetish, as it often is by the laity. Neither makes the milk directly more digestible nor lessens one iota the necessity for proper modification for the infant. The sole purpose of these methods is to kill dangerous germs, and to lengthen the time during which the milk may be safely used as a food. Absolutely fresh, clean milk kept at a low temperature and used with reasonable promptness during the winter months requires no heating. The necessity for pasteurization arises with the slightest uncertainty as to the cleanliness of milk, the healthiness of cows, the delay before consumption, the advent of warm weather, and where milk is to be distributed after modification for use in the homes of the poor, where there is always uncertainty as to its subsequent care. Sterilization of milk is indicated where any serious doubt exists as to its source, when it is to be preserved for a long time, as on a journey or voyage, and, perhaps, also where it is to be distributed in the hot months among the ignorant and careless poor.

**CREAM.**

This term, signifying the more concentrated fatty portion of milk, has led to much confusion, since what passes for cream may contain anywhere from 8 per cent. to 40 per cent., or even more, of butter-fat. Cream raised by the old shallow-pan system contains, when carefully skimmed, about 16 per cent. fat. This and all other creams which rise naturally to the surface, as upon bottled milk, owing to the lesser specific gravity of the fat, are known as gravity creams. Cream so raised undergoes no change of its fat globules, and is to-day esteemed by many authorities as superior for infant feeding to the centrifugal cream removed from milk by the mechanical action of the separator machines, which are thought to disturb the integrity of the fat globules, making them more liable to coalesce. Separator cream may be of almost any density and percentage, depending upon the speed and number of revolutions at which the machine is manipulated. Cream is always richer in bacteria than skimmed milk, since these are mechanically carried along with the fat globules. Cream as sold in the market is often thickened by the addition of substances which swell up the mucoid proteid, and so make it appear richer than it is. It is often, when sold, older than milk marketed the same day. A not uncommon error is to contaminate a fairly fresh milk with an old bacteria-laden cream. It is much better, if it is desired to have more fat than proteids in any modification of milk, to use the upper layers of a milk which has stood a sufficient length of time to have the fat chiefly in the upper portion. This is known as "top milk," and will be referred to as such hereafter. The advantage of the term lies in the fact that it calls attention to the fact that we are simply dealing with an extra fat milk, and that the other elements of the milk are still there in nearly the same proportions, although actually the percentage of proteids decreases progressively to a slight extent as the percentage of fat rises. Since the stronger top milks are necessarily much diluted in infant feeding, this error is reduced; so that for practical purposes it may be disregarded. The visible cream layer which rises upon bottled milk does not vary very much in its height and amount, but is much denser—*i. e.*, contains more fat—in a rich than a poor milk. Moreover, as will be shown later, the visible cream in any bottle is not of the same richness throughout, the action of gravity making the upper portion denser than the lower; so that if dipped off separately the top ounce would contain a much larger percentage of fat than the lowest ounce of the cream layer. It has further been shown that after the cream has risen in a milk bottle which is usually filled entirely full, ordinary handling in transportation does not disturb the percentages in the layers.



## CHAPTER VIII.

### SUBSTITUTE INFANT FEEDING—FEEDING AFTER THE FIRST YEAR.

#### GENERAL PRINCIPLES INVOLVED IN SUBSTITUTE FEEDING.

In the evolution of modern medicine one of the last problems to be attacked and reduced to a scientific basis has been that of substitute infant feeding. This has been largely due to the fact that under normal conditions the human infant received its suitable nourishment ready-made from the maternal breast, and owing to the general cheapness of human life comparatively little attention was paid to that small proportion of infants who must needs fight an often losing fight for existence unless they could be nourished by a foster-mother. However the startling increase in the number of mothers who from physical and social causes cannot nurse their offspring, together with a tendency to smaller families, has, with the constantly increasing value to the individual life, aroused a wider interest in the subject.

In order to understand the principles which at present form the basis of artificial feeding, it is well to be familiar with the various theories which have been held at different times, and which, proving imperfect or fallacious, have at the same time contributed in certain ways to the sum of our experience. Many have been retained in larger or smaller part, but modified in accordance with our expanding knowledge. When the milk of the mother failed, some substitute was necessary, and was chiefly sought by analogy in the milk of other animals. This has finally been narrowed down to that of the cow. But few children could digest this pure; hence dilution was practised. Chemists attempted to prepare foods which could be easily digested, but unless they were combined with milk, and even when so combined, they failed in the main to produce perfect nutrition. Condensed milk obtained wide use, but its low fat and proteids and high sugar content produced fat, flabby growth without resistance to disease. Some knowledge of the chemical composition of breast milk then led to attempts to imitate its average proportions from cows' milk, and the problem seemed solved, but it did not take fully into consideration the inherent differences in the proteids of the two milks, and peptonization (pancreatization) was advanced to pre-digest the excess of casein. Bacteriology then revealed the excessive germ content of milk, and their destruction by sterilization was advocated; but this gave way to pasteurization, and more recently the problem has been attacked at the right source in efforts to secure clean, fresh milk of low bacterial content which need not be altered by cooking. Taking more accurate analyses of breast milk for the basis, success was

then sought by preparing milk which should conform to these analyses in at least containing the proper proportions of fat, sugar, and proteids, and by arranging a schedule of increased strengths which should advance with the age and growth of the child. These were invariably combined with the use of some alkali in the food, which we now know has a definite effect upon the digestive processes. Greater success was attained with normal children than before, but for many others it was soon found necessary to elaborate a plan by which the various elements could be raised and lowered at will. To this end an exact knowledge and control of the contained percentages were necessary, which led to the establishment of milk laboratories in which any proportions determined upon could be produced. These combinations were first prepared from cream and skimmed milk obtained by the use of a centrifugal separator. Then there arose an objection in some quarters to the use of centrifugal cream, and this and the necessity for adapting the method to preparation in the home led to a wider employment of the richer, upper layers produced upon standing by the action of gravity in bottled milk. The chief difficulty has been to secure in every case the proper digestion of a sufficient amount of the peculiar proteids of cows' milk to maintain nutrition. Cereal gruels, which have long been used as diluents, and recently have been more commonly dextrinized, now claim a new place in that they are said to mechanically render the casein coagulum smaller and thus more readily digestible. Egg-albumen has been tried to supplement deficient proteids, but the most recent move of importance, in difficult cases, has been to add the soluble proteids of whey, called loosely "whey proteids," to bring the proteid content up to the needs of the child. We are but just beginning to understand that as the digestive secretions of the child's stomach make their appearance their chemical action upon the milk ingested forms with it combinations which are retained and acted upon longer by the stomach instead of being passed on into the intestines, and that by this means the stomach fits itself in time for the digestion of solid food, but the larger and tougher curds formed from cows' milk seriously complicate the matter. Upon the further elucidation of these problems of digestion lies our greatest hope of progress in the future. In the mean time the opinion has been reached upon all sides that there is no single, royal road to successful feeding in all cases, but that children must be studied as individuals and their food adapted to each, not only with a competent knowledge of various methods and of the indications for their application, but also with a view not alone to their increase of weight, but to their perfect nutrition.

**Food Elements and their Purposes in Nutrition.**—The necessary elements of food to maintain life and to provide for growth and repair are fat, proteids, carbohydrates, mineral salts, and water, and the proportions of these required depend upon the species and the type of the digestive organs. The adult requires these largely for the production of heat and energy and to replace tissue waste. The young demand them in addition for tissue building in their more or less



rapid growth. Each element plays its own distinctive part in the economy.

Fats and carbohydrates containing hydrogen, carbon, and oxygen are producers of heat and energy, which may also be stored up potentially in the body as fat. Proteids, which contain in addition to hydrogen, carbon, and oxygen also nitrogen, sulphur, and phosphorus, are the only true tissue builders.

**Fat.**—Fat not only appears as such in the body, but is necessary for proper building of the nervous and osseous systems. As a fuel for the maintenance of body heat it has two and one-fourth times the value of sugar or proteid, and one of its important functions is to spare the proteid from being drawn upon for heat production. Breast milk contains from 3 to 5 per cent. of fat, and we endeavor during the first three months to give as near 3 per cent. as possible in the food, not alone for the immediate needs of the body, but because a larger percentage of fat than proteid favors mechanically by its presence the digestion of the proteids, while the residue of unabsorbed fat serves to maintain a soft consistency of the feces, preventing constipation.

**Proteids.**—From the foregoing the immense importance of proteids in the food is self-evident, for without their absorption in suitable amounts there can be no proper growth and development, and we can readily understand that, since the young infant must begin, on account of its more difficult digestibility, with proteid percentages of cows' milk much below that of the 1.50 per cent. contained in breast milk, the bottle-fed child is necessarily handicapped from the start until it can digest an amount of proteids equal to that of breast milk, which is rarely the case before the fifth to sixth month. Proteids are also blood builders, and prolonged deficiency of proteids in the food produces anemia as well as malnutrition. Proteids can be called upon to produce body heat, but such a necessity is disastrous and should be prevented by furnishing ample fat and carbohydrates in the food.

**Carbohydrates.**—Carbohydrates, which include sugars and starches, play a most necessary role. They can be and are converted in the body into fat, and are an important source of animal heat, but, like fat, they cannot restore nitrogenous waste nor build new cells.

**Mineral Salts.**—Mineral salts not only are necessary for the formation of bone but of other tissues, and for the secretions of organs which carry on the functions of the body.

**Water.**—This enters largely into the composition of the body, even the bones containing 10 per cent., but is also required to maintain its fluids and the functions of digestion, secretion, and excretion. The major part must be introduced as such with the food. A smaller proportion is released by digestion from mechanical or chemical combination with the food.

All experiments in nutrition have resulted practically in the same conclusion that each animal must have a well-balanced ration suited to its special needs. All deviations from the normal, if persisted in, are eventually productive of harm. The modern feeding of infants is

based upon the principle that breast milk is the ideal food for them, and that any substitute food, to be successful, must resemble it closely, not only by furnishing the same elements, but the same elements in as nearly as possible the same form, and also by maintaining a similar balance in their proportions and producing normal development of the digestive organs. Cows' milk, when properly modified in the proportions of its elements, is the only generally available substitute which can fulfil these conditions with reasonable approximation, since no manufactured food has ever been devised which does this. Nevertheless, when approaching the subject of the modification of cows' milk we must start with the definite understanding that while all milks resemble each other in gross appearances, human milk and cows' milk are in certain other respects two very different fluids designed by nature to meet the needs of the young of two different species with different requirements and different types of digestive apparatus. These differences are best shown in the following table, freely adapted from Rotch:

TABLE COMPARING WOMAN'S MILK AND COWS' MILK.

Reaction.	NUTRITIVE DIFFERENCES.	
	Woman's milk directly from the breast.	Cows' milk as ordinarily received (about twenty-four hours old).
	Faintly acid to phenolphthalein.	Slightly acid to phenolphthalein.
Water.	85.00 to 90.00 per cent.	85.00 to 88.00 per cent.
Mineral matters.	0.18 to 0.25 "	0.70 to 0.75 "
Total solids.	15.00 to 19.00 "	15.00 to 12.00 "
Fats.	3.00 to 5.00 " (relatively poor in fatty acids).	3.00 to 5.00 " (relatively rich in fatty acids).
Lecithin.	larger amount.	smaller amount.
Sugar.	6.00 to 7.00 per cent.	4.00 to 5.00 per cent.
Proteids (total).	1.00 to 2.25 "	3.00 to 4.00 "
Casein.	?	2.00 to 3.00 "
Soluble proteids.	?	0.65 to 0.85 "
	PHYSIOLOGICAL DIFFERENCES.	
	Coagulation of casein by acids.	Coagulation of casein by rennet.
	Curds with difficulty in the flocculi.	Curds easily with small amounts of acids in finely divided curds. With larger amount of acid in larger and more tenacious curds.
	In loose flocculi.	Solid mass.
	Flocculent precipitate, readily digested.	More or less tough curds of various sizes with tendency to shrink. Digestibility varies with size of curd and quantity of gastric secretion.

NOTE.—The methods of separating the casein and soluble proteids of cows' milk are not applicable to human milk because the caseins behave differently with reagents. No analysis thus far made can be accepted without question.

Comparison of the above table not only shows that while the percentage amounts of the fat are the same, those of the sugar, proteids, and mineral matter are quite different. But it is still more necessary that we should understand that there are very vital differences in the composition in some of these groups. The most noticeable difference is in the proteids. In cows' milk there is a marked preponderance of casein over soluble proteids. In breast milk, according to the best recent analyses, the

soluble proteids exceed the casein, although not as largely as was formerly supposed. The difficulty in separating these bodies is greater than in cows' milk, and there is some doubt whether it has as yet been satisfactorily accomplished. The marked contrast between the behavior of the caseins of the two milks with acid and rennet during digestion, which causes the chief difficulty when cows' milk is fed to the infant, is not accounted for by the differences in the quantities, since it is not materially altered by dilution of cows' milk, and points strongly to inherent chemical differences. In the mineral ash of cows' milk, which is more than three times that of breast milk, there is more lime, magnesium, potassium, and phosphoric acid, and less chlorine and sulphur. The lecithin in breast milk, which enters into the formation of the nervous system, considerably exceeds that in cows' milk.

Even the fats, although largely the same, differ because of the presence in cows' milk of large amounts of volatile fatty acids. When these differences in composition and digestibility are considered, together with the probability that breast milk contains properties which nature designed especially for the requirements of the human infant, we will see that no amount of dilution or modification will produce an exact counterpart of breast milk; therefore, the infant so fed must have its digestive tract trained to utilize a different kind of food from that which nature intended. We recognize from the foregoing table that 6 to 7 per cent. of milk-sugar in breast milk can be utilized by the infant, that the fat as supplied by the mother exceeds the amount of proteid, and that the ability to digest a certain percentage of proteid (1.5 to 2 per cent.) should be attained as soon as practicable, and that these elements should be sufficiently diluted with water. Beyond this we are not able to go. The salts, the enzymes, the protective principles, and other properties which we can only surmise that breast milk contains, we as yet make no attempt to imitate. Yet, despite the radical differences between the two milks, much more successful feeding than was formerly attained has been accomplished by modifying the relative proportions of the elements of cows' milk, taking as a general guide to the requirements of the infant the composition and percentages of breast milk. Where failure has occurred it has been largely due to adhering to these too closely in all cases, and losing sight of the still irremediable differences in digestibility.

**Modified Milk.**—Modified milk is primarily any milk which has undergone any change in the amounts or relations of its constituent parts, so that the old-time physician who fed infants on diluted and sweetened cows' milk used a modified milk. As the term is now used, it is ordinarily applied to cows' milk prepared for infants by decreasing or increasing any of its constituent parts, or the addition of other substances which, of course, includes its dilution with water. This is now based upon a clearer knowledge of the probable requirements of the infant, which has come from a study of the analyses of breast milk. From this we learn that cows' milk contains from two to three times as much proteid as breast milk; therefore, we dilute the cows' milk, and,



since this also reduces the amount of fat and sugar, we employ measures to increase these to the requisite amount.

**Percentage Feeding.**—Percentage feeding, so called, is but a further step, and simply consists in making our modifications of cows' milk in such a way that we know approximately the amount of fat, sugar, and proteid in the food, when it is prepared, as an intelligent guide first to its selection for any particular case, and secondly, what is, if anything, more important, as a guide to any subsequent changes which may be found necessary. We are then not working, as of old, in a hap-hazard manner and in the dark, but upon definite known lines, with a rational scientific basis. The common error of seriously disturbing an infant's digestion by jumping from a much diluted condensed milk containing a small percentage of proteids to diluted cows' milk with a high proteid percentage will not be made by one who has studied the subject enough to know the differences and to form a rough working estimate of percentages. Every practitioner should at least understand the meaning of the values which they represent. The ability to think in percentages so that any given dilution of cows' milk, or of a cream of known strength, with any given number of parts of water at once suggests the approximate percentages of fat and proteid can be readily acquired by some study and by practice. It gives to the subject a hitherto unknown interest.

**Calculating Percentages.**—Although we have seen that average cows' milk contains 4 per cent. fat, 5 per cent. sugar, and 3.50 per cent. proteids, since the fat and proteids vary in different milks, we may assume for practical purposes of calculation that these are 4 per cent. fat, 4 per cent. sugar, and 4 per cent. proteids, and one who desires to acquire the habit of rapidly estimating percentages will do well to work at first upon this basis. If it is desired, upon the one hand, to dilute one part of milk with 1, 2, 3 or more parts of the diluent (water, barley-water, etc., as the case may be) the resulting amount of fat, sugar, and proteids will be found by dividing the constant number 4 by the total number of parts of milk and diluent added together. Thus, in 1 part milk and 1 part water divide the 4 per cent. each of fat, sugar, and proteids by the total number of parts, which is 2, and gives 2 per cent. each of fat, sugar, and proteids in the mixture; 1 part of milk and 2 of water divide 4 by 3 and give 1.33 per cent. each of fat, sugar, and proteids; 1 part milk and 7 parts water divide 4 by 8 and give 0.50 per cent. each of fat, sugar, and proteids. The sugar, however, is easily adjusted later, so that we require only to determine the amount of fat and proteids.

On the other hand, if we desire to prepare a mixture for the infant which contains only 0.50 per cent. of fat and proteids and wish to know how many parts of water are required, we divide the 4 per cent. of fat and proteids in 1 part of milk by 0.50 and find that it goes eight times—i. e., the mixture will be one-eighth the strength of plain milk giving the fraction  $\frac{1}{8}$ . Our mixture then would consist of 8 parts—that is, 1 part milk diluted with 7 parts water. Again, if we wish a mixture containing 1.00 per cent. each of fat and proteids we divide 4 by 1, which gives us 4 parts for our mixture or the fraction  $\frac{1}{4}$ , which will

be 1 part milk and 3 parts diluent. The most common error is to consider only the parts of the diluent and neglect the parts of milk. With this one exception the matter is very simple.

Slightly more difficult to grasp is where more than 1 part of milk is used. For example, 2 parts of milk and 1 part of water. Here, as before, we divide by the total number of parts, or 3; but since each part of milk used contains 4 per cent. each of fat and proteids, 2 parts contain 8 per cent. of each, and this 8 per cent. divided by 3 gives us 2.66 per cent. in our mixture. Reversing this process and desiring to form a mixture containing 2.66 per cent. each of fat and proteids—that is, containing two-thirds of the amount of fat and proteids in plain milk (4 per cent.)—we make a mixture of 3 parts, 2 of which will be milk and the remaining 1 part of water.

Were it 1.60 per cent. of fat and proteids which were desired, or two-fifths of the fat and proteids in plain milk, we would require a total of 5 parts, of which 2 parts are milk and the 3 remaining parts water. In short, the fraction shows what proportion of the feeding mixture must be milk, whether it be of one feeding or of a supply sufficient for the entire day. For a single bottle to contain 1.60 per cent. fat and proteids, which would require two-fifths milk, we may use 2 ounces of milk and the remaining three-fifths or 3 ounces diluent. The same quantities may be multiplied by the number of feedings for the day; or, if each bottle is to contain more or less than 5 ounces, we may make up 10, 20, 30, or 40 ounces, of which two-fifths are milk and the remaining three-fifths diluent, place the exact amount desired in each bottle, and reject any excess.

As familiarity with this process develops, one comes to associate the percentages most commonly employed with the fraction which represents their relation to plain milk. One-half hour spent with pencil and paper in verifying each step in the above figures and those in the following table will prove more useful in mastering the principles than many readings of the text.

TABLE SHOWING THE NUMBER OF PARTS OF COWS' MILK AND DILUENT REQUIRED TO SECURE CERTAIN DESIRED PERCENTAGES OF FAT AND PROTEIDS.

Percent. of fat and proteids desired.	Fraction repre- senting amount of milk in mixture.	Total parts.		Parts milk required.	Parts diluent required.
0.50	$\frac{1}{10}$	10	less	1	leaves 9
0.60	$\frac{1}{12}$	12	"	1	" 11
0.80	$\frac{1}{6}$	6	"	1	" 5
0.90	$\frac{1}{7}$	7	"	1	" 6
0.95	$\frac{1}{8}$	8	"	1	" 7
1.00	$\frac{1}{4}$	4	"	1	" 3
1.15	$\frac{1}{5}$	5	"	1	" 4
1.33	$\frac{1}{3}$	3	"	1	" 2
1.60	$\frac{2}{5}$	5	"	2	" 3
2.00	$\frac{1}{2}$	2	"	1	" 1
2.50	$\frac{3}{5}$	3	"	2	" 1
3.00	$\frac{3}{4}$	4	"	3	" 1



Our primary purpose in diluting cows' milk is to reduce the excessive amount of proteids, not, as used to be taught, to make it correspond to that of an average breast milk, but to a point where the dissimilar proteids will be readily digested by the infant. By such dilutions, carried as far as may be deemed advisable, the percentage of proteids in cows' milk may be reduced to any point determined upon as suitable for the individual infant, decided by its digestive ability, but in so doing the percentages of fat and sugar will also be lowered. How, then, may the fat and sugar be raised or secured in the mixture in suitable amounts?

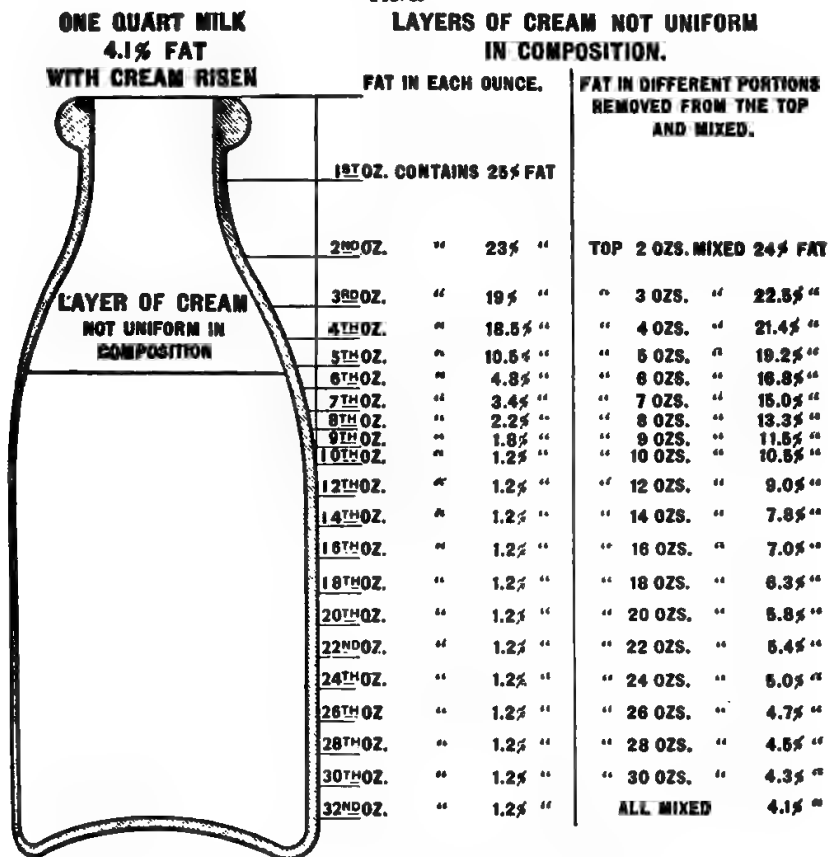
**To Secure the Required Proportion of Fat in Modified Milk.**—Experience has shown us that older methods, which call for the adding of cream to milk to raise the amount of fat in the mixture, are not only exceedingly inaccurate in their results, owing to great variations in the strength of so-called "cream," but also require too complicated calculation on the part of the physician, beside increasing the dangers arising from previous manipulations of the ingredients. (See page 122.) The system, therefore, which bids fair to supplant all others is that of the use of "top milks" of various strengths, to furnish the different percentages of fat which we may require. Fortunately, also, this system is equally applicable to cities and large towns, where milk bottled at the farm should always be obtainable, and to the country, where it may be placed in quart preserving jars soon after milking. Careful analyses have been made of each successive ounce removed from the top downward from the ordinary quart milk bottle, and have shown that after standing until the cream has risen the top ounce is richest in fat and the lowest the poorest in fat for the upper ten ounces, with a progressive decrease from the first to the tenth. This shows that not only has the visible cream layer, which amounts on the average to between 5 and 6 ounces, a different density in each successive layer, but that the same is true of the upper part of the milk upon which the cream has risen. This will be shown by the first column of the table on page 131, taken from Chapin (Fig. 29).

Below the tenth ounce the skimmed milk is assumed for practical purposes to have about the same fat content. By inspection of the second column of figures, in the same table, it will be readily seen that when 2, 3, or more ounces are removed from such a bottle, and mixed, each additional ounce reduces the percentage of fat in the mixture, since it is progressively diluted by those containing less and less fat. To avoid misunderstanding it must be stated at this point that the amount of fat contained in any given number of ounces of top milk is not the same with poor, average, and rich milk, although the ratio of fat to proteids remains about the same. Therefore, if the milk is very rich, 5 per cent. fat, or very poor, 3 per cent. fat, about 2 ounces more and 2 ounces less, respectively, should be taken to get suitable percentages of fat. A good average milk which contains about 4 per cent. fat is preferable for infant feeding.

Accuracy in removing the requisite number of ounces is essential to the finer application of these principles. Pouring off the upper portion which is to be used into a graduated measure, or siphoning away the

lower part which is to be rejected, are crude methods which do not permit of great accuracy, although permissible when other means are not available. Various flat, pointed, and round-bottomed dippers have been devised for this purpose, each of which contains 1 ounce, and at the same time serves to remove the upper layers without undue disturbance and also to measure the quantity. A milk bottle properly prepared for shipping is completely full. The first ounce or dipperful must therefore

FIG. 29



Distribution of fat in bottled milk after cream has risen. (Chapin.)

be removed with a teaspoon, after which the other dipperfuls are secured by just submerging the upper edge of the dipper, which skims off the successive layers (Fig. 30). Such dippers are of great value in impressing upon the mother the importance of exact proportions in the infant's food, and may be used by her also for measuring the diluent.<sup>1</sup> By this simple means of removing and mixing different numbers of

<sup>1</sup> A good tinned dipper may be had by mail for ten cents from the Cerco Company, Tappan, N. Y., or an aluminum one for twenty cents from J. Dougherty, 409 W. 59th Street, New York City.

ounces we may then easily secure a top milk containing almost any desired percentage of extra-fat milk. Now, for all practical purposes, the presence of extra fat does not displace a very appreciable amount of proteids; so that we may consider that the percentage of proteids in any

FIG. 30



Chapin dipper for removing top milk; holds exactly one ounce.

given number of ounces removed and mixed, remains the same as that of plain milk, which we know to be 3.50 per cent. to 4 per cent., and we can also secure, by removing and mixing a given number of ounces, any desired ratio between the fat and proteids. This may be 16 per cent., 12 per cent., 10 per cent., 8 per cent., or 6 per cent. fat in the top milk, and consequently 4, 3,  $2\frac{1}{2}$ , 2 or  $1\frac{1}{2}$  times as much fat as proteids, while by shaking the whole bottle so as to redistribute the cream evenly we again secure plain milk in which the fat and proteids are equal or both about 4 per cent. To reduce this principle to a simple working basis it is only necessary for the practitioner to remember the following figures for good average milk, containing about 4 per cent. fat: Top 9 ounces (upper third) gives 12 per cent. fat, or fat three times the proteids. Top 15 ounces (upper half) gives 8 per cent. fat, or fat twice the proteids. Plain milk (whole bottle) gives 4 per cent. fat, or fat equal the proteids. For very rich milk (5 per cent.) take 2 ounces more top milk, and for very poor milk take 2 ounces less.

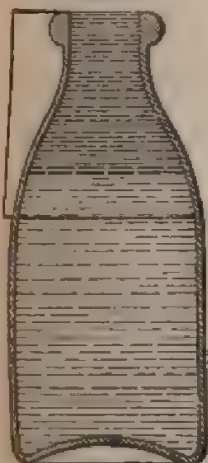
This certainly requires no great effort of memory. The diagram on page 133 will show this in another way. These three furnish the basis for the usual modifications required in the ordinary infant feeding, and the physician who employs these understandingly for a time soon finds himself forming intelligently other combinations of fat and proteids, by removing progressively a larger or smaller number of ounces from the top of the bottle, as he wishes to decrease or increase the proportion of fat to the proteids, since, as may be seen by reference to Fig. 29, taking off less ounces gives a top milk richer in fat, and taking off more ounces one containing less fat.

The same rules are applied to the finding of fat percentages for "top milks" that we have outlined for plain milk (page 128). Thus, with 1 part 12 per cent. fat top milk (top 9 ounces) and 3 parts of diluent we divide the 12 per cent. fat and 4 per cent. proteids by 4, the total parts used, which gives us 3 per cent. fat and 1 per cent. proteids in the mixture. Again, with 1 part 8 per cent. fat top milk (top 15 ounces) and 2 parts of diluent we divide 8 per cent. fat and 4 per cent. proteids by 3, the total parts, and find 2.66 per cent. fat and 1.33 per cent. proteids in the mixture. Having learned from practice what the resulting percentage of the proteids will be if the milk is one-half, one-third, or one-fourth, etc., of the mixture, and knowing that the fat percentage will remain equal to or twice or three times the proteid percentage, whatever

the dilution, according to the strength of the top milk, or milk we are using, the calculation is rapidly made in one's head; then percentage feeding loses its terrors, and we estimate percentages as easily as we would the number of grains to the teaspoonful in a 4-ounce mixture.

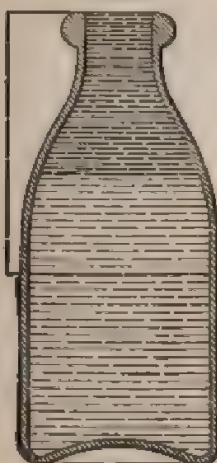
**To Secure Suitable Percentages of Sugar in Modified Milk.**—Breast milk contains 6 to 7 per cent. of milk-sugar, lactose, which varies little in amount throughout lactation. This indicates to us that the infant can absorb and utilize this heat and fat-producing element of its food in considerable amount. Cows' milk contains actually about 5 per cent. lactose, which is naturally still further reduced by dilution of the milk. Since we take breast milk so far as possible as our model, this deficiency must be made up in the infant's food, because not only is it absorbed

FIG. 31



Upper third. Nine ounces top milk. Fat percentage three times that of original milk. Protein percentage same as original milk. Fat three times the proteids.

FIG. 32



Upper half. Fifteen ounces top milk. Fat percentage twice that of original milk. Protein percentage same as original milk. Fat twice the proteids.

FIG. 33



Contents of entire bottle mixed. Plain milk. Fat same as proteids.

with less trouble than the other elements of the food, but because its consumption in the body, like that of the fat, prevents the proteids from being called upon to produce heat when they should be utilized for tissue building. Milk-sugar is preferred by those who adhere closely to breast milk as a model, but cane-sugar may often replace milk-sugar with good results when employed in rather less quantity, since it is both sweeter and more liable to fermentation during digestion. The malt-sugars which enter largely into some infant foods are readily absorbed and are more laxative in their effect. In modifying milk we no longer consider it imperative to calculate the percentage of sugar resulting from the necessary dilution, since, for practical purposes, the addition of a suitable quantity of sugar can be accomplished by rule of thumb. Two and a half fairly level tablespoonfuls of milk-sugar and two exactly level



tablespoonfuls of granulated cane-sugar equal 1 ounce. If 1 ounce of sugar is added to each 20 ounces of milk during modification we shall have, with whatever sugar is already in the diluted milk, about  $5\frac{1}{2}$  per cent. for the weaker formulæ and  $6\frac{1}{2}$  per cent. for the stronger formulæ, which will be about right for our purposes.

In making smaller quantities of food, one fairly level tablespoonful of milk-sugar and one heaping teaspoonful of cane-sugar to 8 ounces furnish the same proportions. When dextrinized gruels are used as a diluent the change in them of the starch to dextrose and maltose calls for making the above measures scant or the total percentages of sugar will be too high. Also, when the milk in the mixture exceeds one-half of the whole the sugar should be gradually reduced to three-fourths and later to one-half the above quantities, especially with the use of gruel diluents. When plain milk is reached no sugar need be added.

**Diluents.**—Starting with the premise that the proteids of cows' milk, as has been shown, are different from those of breast milk, both in amount and in the preponderance of the curd-forming casein, and that this casein of cows' milk tends to coagulate in the stomach in large and tough masses, instead of small, soft flocculi, various methods of preparing the milk have been adopted, at first quite empirically, to overcome this serious difficulty, and each has proven reasonably successful in the hands of the originators and their group of disciples, giving rise, as it were, to several schools of infant feeding.

The attempts to explain scientifically the good results of these empirical methods have not always been based upon correct premises, but the explanations, however, have not impaired the efficiency of the methods when intelligently applied, although they have added materially to the seeming confusion and misunderstanding which surround this subject. The methods are now practically confined to two:

1. Dilution of the milk with water and the addition of lime-water or bicarbonate of soda—*i. e.*, dilution with alkaline solutions.
2. Dilution with cereal gruels, which are frequently dextrinized.

Both reduce by dilution the amount of casein in the mixture. Both also favor curdling in smaller and softer flocculi, but each influences the digestion of the casein in its own peculiar way.

**Alkaline Diluents.**—The addition of alkalies was at first recommended solely with the idea that they made acid cows' milk conform more closely to a supposedly alkaline breast milk. Since the complete demonstration that both breast milk and cows' milk are acid, the original argument for the use of alkalies has lost its force, and, unless some other good reason could be found for its continuation, such use would be irrational. Experience has, however, seemed to show that the addition of such alkalies was essential to the successful feeding with cows' milk when water was used as the diluent, and the explanation is to be found in the fact that, aside from any special effects which each may have upon the curd, alkalies tend to retard or inhibit the action of the rennet ferment of the stomach upon the casein. The alkalies have also more or less effect as antacids which not only neutralize any lactic acid which may



have been formed in the milk, but also combine to some extent with the acids of the stomach, preventing to a greater or lesser degree their forming tough curds with the paracasein, so that less digestion will be required on the part of the stomach.

The action, then, of alkaline diluents is a chemical one. Dilution of milk with cereal gruel is, on the other hand, mechanical. The gelatinous properties of the cooked cereal and its particles of cellulose envelope the flocculi of precipitated casein, preventing their tendency to coalesce into dense masses, and thus allowing more complete penetration of the digestive juices.

**Cereal Diluents.**—It has been urged against the cereal diluents that starch is not an ingredient of breast milk and that the starch-transforming functions of the infant are not fully developed; but we must accept at the outset that cows' milk, however manipulated, will always differ from breast milk, and the dextrinization of gruels, which is now almost universally used for young infants, supplements the action of the developing salivary and pancreatic functions in preparing the starch for absorption; so that the addition of cereals which enables the stomach to digest more casein and so to develop by the exercise of its normal functions, is both justifiable and proper.

Excellent results are obtainable by both methods, and where either one fails the other may be successful. We should, therefore, take no partisan position, but be familiar with the use of each.

**Lime-water as the Alkaline Addition to the Diluent.**—The usually accepted percentage of lime-water in the food of a normal infant is 1 per cent. This is secured by the use of 1 ounce of lime-water in every 30 ounces of food prepared, which would be  $1\frac{1}{2}$  ounces for 30 ounces of food, 2 ounces for 40 ounces, and  $2\frac{1}{2}$  ounces for 50 ounces. In writing directions for the preparation of mixtures, the amount of lime-water must, of course, be subtracted from the total amount of diluent required. For example, if in a 20-ounce mixture there are to be 5 ounces of top milk and 15 ounces of diluent, we write for 5 ounces top milk, 1 ounce lime-water, and 14 ounces boiled water.

The properties of lime-water are mainly those of an alkali; its antacid value is small. In common with other true alkalies it has the property of swelling the mucoid proteid of milk, thickening it, and making a visible change in its consistency. It therefore has a definite effect upon the precipitation of casein, favoring greater flocculence of the masses, and consequently rendering them more readily attacked and penetrated by the digestive juices. Its second and probably its chief influence upon the digestion of milk consists in its effect as an alkali in retarding the clotting action of the rennet enzyme of the stomach upon the casein of milk, but, being a weak antacid, it is soon neutralized by any acid present. The formation of the paracasein clot is slower in the presence of an alkali and cannot take place in a fully alkaline medium until the alkali has been neutralized or removed. Immediate clotting of the milk in the stomach into large masses which are soon transformed by acid into firm curds is therefore interfered with by the addition of lime-water. The

degree of this interference depends upon the proportion of the lime-water to the amount of the *milk* contained in the mixture. Five per cent. of lime-water as ordinarily used in food mixtures probably serves only to make the curds smaller and so more digestible and possibly to delay moderately the clotting by rennet.

Cows' milk is rendered definitely alkaline to phenolphthalein by 60 to 100 per cent. of lime-water, or nearly ounce for ounce. Inspection of the following table will show that the addition of 10 per cent. of lime-water, often recommended for use in the feeding mixtures of young infants, gives a percentage of from 60 to 200 per cent. of lime-water to the milk in the weaker formulæ.

TABLE SHOWING ACTUAL AMOUNT OF BICARBONATE OF SODA OR THE PERCENTAGE OF LIME-WATER TO THE MILK IN MIXTURES MADE WITH TWO GRAINS OF BICARBONATE OF SODA TO THE OUNCE OF MIXTURE, OR 10 PER CENT. LIME-WATER.

Water.	Milk.	Lime-water.	Bicarbonate of soda.	Food.	Per cent. lime water to milk.	Grains of bicarbonate of soda to each ounce of milk.
17 oz.	+ 1 oz.	+ 2 oz.	or 40 grs.	+ 20 oz.	200 %	40 gra. to each oz. milk.
16 "	+ 2 "	+ 2 "	" 40 "	20 "	100 "	20 " " "
15 "	+ 3 "	+ 2 "	" 40 "	20 "	66 $\frac{2}{3}$ "	13 " " "
14 "	+ 4 "	+ 2 "	" 40 "	20 "	50 "	10 " " "
13 "	+ 5 "	+ 2 "	" 40 "	20 "	40 "	8 " " "
12 "	+ 6 "	+ 2 "	" 40 "	20 "	33 $\frac{1}{3}$ "	7 " " "
11 "	+ 7 "	+ 2 "	" 40 "	20 "	28 "	6 " " "
10 "	+ 8 "	+ 2 "	" 40 "	20 "	25 "	5 " " "
9 "	+ 9 "	+ 2 "	" 40 "	20 "	10 "	2 " " "

NOTE.—The use of one ounce of lime-water in twenty ounces (5 per cent. of lime-water) or one grain of sodium bicarbonate to each ounce of the mixture gives one-half of the figures in the last two columns.

The employment, then, of 10 per cent. lime-water in the food of young infants containing only a small quantity of milk amounts to giving them a highly alkalized milk, which will be but slowly, and possibly even not at all, clotted and curdled by the secretions of the stomach, allowing some of the work of digestion to fall upon the intestine, which is at this time better fitted for the purpose. The degree of alkalization with its restraining influence upon gastric digestion is gradually lessened as the child takes stronger mixtures containing more milk. The large percentage of lime-water (10 per cent.) should be reduced as soon as practicable in order that the necessary development of the functions of the stomach should not be unduly retarded. Lime-water is also a useful addition to the food where there is a tendency to vomiting with or without loose movements.

**Bicarbonate of Soda as an Alkaline Addition to the Diluent.**—Bicarbonate of soda (baking soda) is less commonly used than lime-water. The usual recommendation is to add it in the proportion of 1 grain to each ounce of food. This for each 16 to 20 ounces is one-fourth of a level teaspoonful, or as large a pinch as can be taken up between the thumb and forefinger. The impression prevails that 20 and 40 grains of sodium bicarbonate in 20 ounces of food mixture are the exact equivalents in their effects of

1 and 2 ounces (5 per cent. and 10 per cent.) of lime-water in 20 ounces of food mixture. This is in certain important respects an error. Chemically pure sodium bicarbonate, if obtainable, would be an antacid only. Such alkalinity as the usual sodium bicarbonate possesses is due to impurities, owing to loss of carbonic acid gas and the reduction of some of the bicarbonate to the carbonate of soda (washing soda), which is an alkali. This was recognized by the United States Pharmacopeia, which allowed 1 per cent. impurity in the "purified" and 5 per cent. in the "commercial" article. The alkalinity in solutions increases by standing, agitation, or increase of temperature above 59° F. Boiling converts it all into carbonate. Bicarbonate of soda, therefore, reverses the properties of lime-water and is a strong antacid and a more or less weak alkali. It does not swell the mucoid proteid of milk as does lime-water, but if the carbonic acid gas in the salt has not been driven off by previous heating of the food the gas is liberated during digestion, when the salt meets with acid, making the curd more porous. Pasteurizing or sterilizing the food converts more or less of the bicarbonate into the stronger alkaline carbonate. Aside from their respective effects upon the mucoid proteid and the porosity of the curd, lime-water, and bicarbonate of soda differ chiefly in their antacid qualities. As ordinarily employed in the amounts of 20 grains sodium bicarbonate or 1 ounce lime-water to 20 ounces of food mixture, their effects as alkalies upon the retardation of the rennet ferment is about the same, but to secure this amount of alkalinity a much greater amount of antacid has been introduced in the sodium bicarbonate; 1 ounce lime-water would be neutralized by somewhat less than 1 ounce of adult gastric juice of 0.2 acidity (HCl); 20 grains sodium bicarbonate require to neutralize them about 10 ounces of the same gastric juice. It is evident, then, that a longer time must elapse, during digestion, with sodium bicarbonate than with lime-water before an acid reaction can be established in the stomach. The formation of acid curds of paracasein and their digestion by pepsin are longer delayed, and part of the fluid milk escapes into the intestine, lightening the burden of stomach digestion. Double quantities sometimes recommended for young infants—*i. e.*, 2 grains to each ounce—would probably cut out stomach digestion entirely. For this reason sodium bicarbonate often serves us better with children of difficult digestion. It is easier to use among the poor, since it is always at hand, and, being added in a dry form and dissolved in the diluent, does not complicate the directions as does lime-water. It may also be chosen when it is deemed advisable to combine the use of an alkaline antacid with that of a cereal diluent.

**Cereal Diluents.**—These are commonly made of barley, wheat, or oats, although rice and arrow-root may be used in certain conditions. Barley is more commonly chosen for infants under seven months of age or in any tendency to relaxed bowels. Oatmeal contains more tissue-building material than barley and may be selected where the digestion is not disturbed or where there is constipation. Unless contraindicated it is preferable for older infants. Wheat flour is possibly less palatable, non-



laxative, of average digestibility, and of higher nutritive properties. It is used both for younger and older infants. Cereal gruels are employed both plain and dextrinized. Plain gruels should be made with less cereal for young infants, since they contain unchanged starch. They may be made with more cereal when added in relatively small amounts to the milk of older infants, since the resulting jelly will be thinned sufficiently by the milk and the starch-digesting functions are then further developed. Dextrinization converts the raw starch into soluble carbohydrates, and in so doing thins the gruel. It is growing in favor with those who use cereal diluents and is especially adapted to use for young infants whose power of digesting starches is at best very slight. It is best discontinued when the amylolytic function develops toward the end of the first year. In acute vomiting, dextrinized gruels are often retained when even water is rejected. Plain cereal waters and cereal jellies are made by using different quantities of the cereals with the same amount of water. Either prepared flour or the grains may be used, but the latter require at least three hours boiling.

**Cereal Waters.**—Cereal waters are made most easily by using the prepared flours: Robinson's Patent Barley Flour, the barley flour and oatmeal flour of the Health Food Company, New York; Hubbell's Prepared Wheat Flour, and Imperial Granum. All of these have been partially prepared by heat, but the length of time recommended for cooking in their directions is best exceeded. Ordinary wheat flour, rice flour, or arrow-root may also be used. To make such a water (thin gruel), stir one heaping teaspoonful of the flour into a little cold water until no lumps remain. Add this to one pint of boiling water and cook at least twenty minutes, preferably in a double boiler (Fig. 26), stirring constantly. One or two ounces of the water will boil away and this may be replaced. Add a pinch of salt and strain through a wire strainer to remove coarse particles.

**Cereal Jellies.**—These require one heaping tablespoonful of flour to a pint of water, but are otherwise made the same way.

By the use of a good type of steam-cooked, flaked or rolled oats, an oatmeal-water or jelly may be made by using double the quantities mentioned for the flour and the same amount of water, and boiling thirty minutes, straining, etc.

**Dextrinized Gruels.**—Depending upon the age of the child and the quantity to be used in the food, one heaping teaspoonful to one heaping tablespoonful is cooked as above in one pint of boiling water for fifteen to twenty minutes. The dish is then set in cold water until the contents are just cool enough to be tasted when the dextrinizing agent is added; stir and keep warm until the gruel becomes thin, after which add a pinch of salt, strain and cool. Various preparations of diastase may be used for this purpose, but that chiefly employed by those who advocate dextrinization is a glycerinated solution of diastase under the name of Cereo, of which thirty drops suffice to convert a pint of gruel. A sufficient quantity of gruel for twenty-four hours' use should be made freshly each day.

All cereal diluents must either be cool before they are mixed with the milk or, if added warm, the food must be quickly cooled, since warmth favors bacterial growth in the milk. If the milk is to be heated at all, as in summer, it will blend better with certain of the plain gruels when it is added to them while they are still hot, in which case the mixture should again be heated to just short of the boiling point, stand twenty minutes for pasteurizing, and then be promptly cooled.

#### PREPARATION OF THE INFANT'S FOOD.

In the city it is essential to secure "certified milk" or a good, clean, fresh milk from a known source, bottled at the farm. In the country completely fill with fresh strained milk a clean quart milk bottle or quart preserving jar, seal, and set in ice or in cool running water overnight, or for at least four hours. Make this period the same each day in order that the cream may rise to the same extent. Remove with a Chapin dipper or pour from the top the requisite number of ounces of top milk to secure the desired ratio of fat to proteids and place in an absolutely clean bowl, glass pitcher, or graduate, and devote the remainder of the milk to other household purposes. Of this top milk so removed, again measure the desired number of ounces (dippers) needed for the feeding for twenty-four hours. Add to this the measured amount of boiled water, lime-water (or bicarbonate of soda), or of the cereal gruel determined upon as the amount of the diluent. Dissolve the sugar in the water. Have as many bottles as there will be feedings in the twenty-four hours. Mix and pour into each of these the amount required for a single feeding. Stopper with clean absorbent or baked non-absorbent cotton, pasteurize, and cool in running water if necessary. Finally place the bottles on ice or keep them at a temperature below 50° F. Immediately before the feeding time remove the cotton stopper, adjust the nipple, heat to blood heat by placing in moderately hot water, test the temperature by allowing a few drops to fall from the nipple upon the inner surface of the wrist, and, after feeding, reject any milk which may remain. Never make a double quantity in one bottle, never feed a second time from a bottle which has once been warmed up.

If, as frequently happens among the poor, it be impossible to secure so much attention to detail, considerable security may be obtained by having the daily supply of food, when mixed, placed in a saucepan and brought up to a point just short of boiling, allowed to stand covered twenty minutes, and then poured into well-scalded quart milk bottles or preserving jars, which are then sealed, rapidly cooled in running water, and, when cooled, kept on ice or in the coolest place available. Although there are opportunities for contamination from repeated opening of such bottles to remove the portions required for each feeding, there will, if the bottle is shaken each time, be greater uniformity in the food and vastly greater protection from contamination owing to the heating of the milk soon after its receipt than is usually the case when the food is



mixed for each feeding from materials which have been more or less exposed throughout the day. Milk should not be kept in an open vessel, even in a refrigerator, since it readily absorbs odors and noxious qualities. Much less should it stand uncovered in a room or on a window-sill, exposed to dust.

**Choice and Care of Bottles.**—Tall cylindrical bottles with a sufficiently wide neck to allow of easy cleansing are preferable to other shapes and suited for use in the ordinary forms of pasteurizers. The markings upon such bottles are more nearly accurate than upon other shapes. Small bottles are made, but those containing 8 ounces serve until the end of the first year, when larger ones may be substituted. After feeding, the bottle should be rinsed free from all vestiges of milk with cold water and then rinsed in scalding-hot water and inverted to dry. Before again using to make up the daily supply of food, they may be boiled; but if previously well cleaned as directed, rinsing in hot boiled water will suffice.

**Choice and Care of Nipples.**—Those made of black rubber are the best. It should be possible to turn them inside out when cleansing. They should fit directly upon the neck of the bottle and have no complicated valves or tubing. The single perforation in the tip should allow the milk to drop when the bottle is turned down, for if it runs in a stream the infant will take the food too quickly. As soon as the feeding is finished the nipple should be washed carefully inside and out with cold water, then in hot water, and placed in a cup of water containing a large pinch of borax or bicarbonate of soda. So cared for, boiling, which softens the rubber, should be only exceptionally necessary. Two nipples may be used alternately and renewed from time to time. Nipples that are cracked or where the hole is large should be discarded.

**Bottle Cosies.**—For young and delicate infants and in cool weather, especially for those who take their food slowly, it is often well to provide small flannel or cotton-flannel bags to slip over the bottle closely and tie about the neck with a drawstring. These aid in maintaining the proper temperature in the food, which otherwise may cool rapidly before it is all taken. Undue cooling may either disturb digestion or cause the child to refuse the full amount. Such bags should be kept scrupulously clean by frequent washing, as they become wet with the food, which soon sours. On the other hand, food should never be warmed except for immediate use. Food warmers which keep the food warm several hours at night, to save lazy and ignorant nurses or parents from getting out of bed, are simply incubators for developing swarms of bacteria even in pasteurized and sterilized milk, and are often the cause of much serious disturbance.

#### DETERMINATION OF FOOD PROPORTIONS.

To secure feeding formulae, tables are frequently printed giving the exact proportions of milk, water, sugar, etc., required to produce a

definite number of ounces of certain fat and proteid ratios presumed adapted to the infant during given periods of its existence. These, while undeniably of assistance to the physician whose sole wish is to secure most easily a food prescription for a baby, tend directly to perpetuate the fallacy that a child of so many months will or should be able to digest the particular formulæ outlined. To secure any real degree of success in feeding infants, which only comes with the ready ability to vary the proportion of each ingredient of the formulæ, the practitioner must learn to make his own formulæ as he must learn to write his own medical prescriptions. The most concise aid for this is the following table freely adapted from that of Dr. J. F. Connors:

KEY TO HOME MODIFICATION OF BOTTLED MILK.

Proteins used in most infant calculations. In skimmed, whole, or top milk, 4 % proteids. Figures showing actual proteid percentages in milk, fat, or top milk, 3.5 % proteids.	Per cent. fat.								Per ct. sugar.	Proportions of milk and diluent in feeding mixtures.				
	Skimmed milk, fat about 1 %.									For skimmed, whole, or top milk, 5 %.	Proportions of milk.			
	Good whole milk, fat about 4 %.										Parts milk or top milk.	Parts diluent.		Total parts.
	Top, 20 ozs., fat 5 %, 1½ times whole milk.													
	Top, 15 ozs., fat 6 ⅔ %, 2 times whole milk.													
	Top, 11 ozs., fat 10 %, 2½ times whole milk.													
	Top, 9 ozs., fat 12 %, 3 times whole milk.													
	Top, 8 ozs., fat 14 ⅔ %, 3½ times whole milk.													
	Top, 7 ozs., fat 16 %, 4 times whole milk.													
0.5	0.23	0.06	0.25	0.37	0.50	0.62	0.75	0.87	1.00	0.31	1/16	1	15	16
0.5	0.29	0.08	0.33	0.50	0.67	0.83	1.00	1.17	1.33	0.42	1/12	1	11	12
0.5	0.44	0.13	0.50	0.75	1.00	1.25	1.50	1.75	2.00	0.62	1/8	1	7	8
0.5	0.60	0.14	0.57	0.86	1.14	1.43	1.71	2.00	2.30	0.70	1/7	1	6	7
0.5	0.60	0.17	0.67	1.00	1.33	1.67	2.00	2.34	2.67	0.83	1/6	1	5	6
0.5	0.70	0.20	0.86	1.20	1.60	2.00	2.40	2.80	3.20	1.00	1/5	1	4	5
1.00	0.90	0.25	1.00	1.50	2.00	2.50	3.00	3.50	4.00	1.25	1/4	1	3	4
1.14	1.00	0.29	1.14	1.70	2.30	2.85	3.40	4.00	4.60	1.43	2/7	1	2½	3½
1.23	1.16	0.33	1.33	2.00	2.67	3.33	4.00	4.66	5.33	1.60	1/3	1	2	3
1.60	1.40	0.40	1.60	2.40	3.20	4.00	4.80	5.60	6.40	2.00	2/5	2	3	5
2.00	1.75	0.50	2.00	3.00	4.00	5.00	6.00	7.00	8.00	2.50	1/2	1	1	2
2.50	2.20	0.62	2.50	3.75	5.00	6.25	7.50	8.75	10.00	3.12	5/8	5	3	8
2.67	2.33	0.67	2.67	4.00	5.33	6.67	8.00	9.33	10.67	3.53	2/3	2	1	3
3.00	2.62	0.75	3.00	4.50	6.00	7.50	9.00	10.50	12.00	3.75	3/4	3	1	4
3.20	2.80	0.80	3.20	4.80	6.40	8.00	9.60	11.20	12.80	4.00	4/5	4	1	5

NOTE.—The proteids have been calculated upon the basis of both 4 per cent. and 3.50 per cent. The former, 4 per cent., is for those who use round numbers to facilitate mental calculation of percentages. The latter, 3.50 per cent., which is the actual percentage of proteids in good average milk having 4 per cent. fat, is to enable the practitioner to determine readily the more exact amount of proteids in any given

mixture. Either column may be used for the purpose of making a mixture of any desired percentages, or in determining the percentages contained in any mixture of known proportions. To make up any desired percentage mixture (1) find in the one of the proteid columns determined upon the desired percentage, or that which is nearest to it; (2) move in a horizontal line to the right until the desired percentage of fat is reached, or one which is nearest to it; (3) the heading of this fat column tells what kind of milk is to be used; (4) on the same line with the fat percentage at the right will be found the fraction showing the necessary proportions of this milk or top milk in the food mixtures to give the percentages selected, and beyond this will be found the number of parts of such milk or top milk and of diluent (see p. 134) which must be used; (5) dip off the proper milk and dilute all or a part of it, depending on the quantity of the food to be made up; (6) the addition of 2½ fairly level tablespoonfuls of milk-sugar or 2 exactly level tablespoonfuls of granulated sugar for about every 20 ounces of the total mixture (see p. 133) will give the proper percentages of sugar.

Of almost equal importance to the selection of proper proportions for the infant's food is the giving of the food in proper amounts and at proper intervals.

QUANTITY OF FOOD.

Authorities agree that the capacity of the stomach of the newborn infant of average weight is about one ounce. Under the stimulus of its newly assumed functions the stomach develops rapidly during the first three or four months, the period during which, under normal conditions, the increase of body weight is also the most rapid. During the fifth to sixth months the rate of increase of both stomach capacity and weight is distinctly less, but it thereafter again increases, although more slowly than in the early months. Attempts to estimate average capacities at different periods necessarily give varying results, owing to the different methods employed and the conditions under which the estimates are made. But although these results show considerable latitude, they are sufficient to allow of the construction of a table for our guidance.

SCHEDULE FOR AN AVERAGE HEALTHY INFANT, SHOWING QUANTITIES, NUMBER, AND INTERVALS OF FEEDINGS. (HOLT.)

Age.	Quantity for one feeding. Ounces.	Number feedings in twenty-four hours.	Interval by day.
Premature infants . . .	1¼ to 3¼	12 to 18	1 to 1½ hours.
First to fourth day . . .	1 " 1½	6 " 10	2 " 4 "
Fifth to seventh day . . .	1 " 2	10	2 "
Second week . . .	2 " 2½	10	2 "
Third week . . .	2 " 3½	10	2 "
Fourth to eighth week . . .	2½ " 4	9	2½ "
Third month . . .	3 " 5	8	2½ "
Fourth month . . .	3½ " 5½	7	3 "
Fifth month . . .	4 " 6	7	3 "
Sixth to tenth month . . .	5 " 8	6	3 "
Eleventh month . . .	6 " 9	5	4 "
Twelfth month . . .	7 " 9	5	4 "
Thirteenth month . . .	7 " 10	5	4 "

Nursed infants of the same age often take and often can only secure very different amounts from the breast. But the thorough utilization of breast milk in digestion, and the more concentrated nature of the maternal milk, which leads sooner to satiety as compared with the weak modifications of cows' milk a young infant can digest, render the adjustment between capacity and supply more automatic than in bottle feeding, so that the danger of overfilling the stomach is minimized. This danger, on the contrary, is a very real one in bottle feeding. An overfilled stomach cannot properly carry on its secretive, digestive, and mechanical functions. Much failure in infant feeding is due to this cause alone, even when other conditions are such as to favor success. It is so common an error that the danger should always be kept in mind. Although bottle-fed infants probably require rather more of their necessarily dilute food, more perfect digestion will undoubtedly be secured by keeping the amount very close to that taken by the nursing infant. But since the rate of body growth and the size of the stomach show a certain degree of relation, the published table allows moderate latitude for different children at the same age. However, unless the rapid growth of the infant distinctly indicates an approach to the larger amount, conservatism will lead to the adoption of the smaller amounts or, at most, of a middle course.

#### THE INTERVAL OF FEEDING.

The establishment of definite hours of feeding contributes largely to success. (See schedule, p. 142.) Irregularity has even more of a pernicious effect upon the infant than upon the adult. Although in bottle feeding lack of uniformity in the strength of the food does not play a part as it does when the breast is given to the infant at irregular intervals, still bottle feedings should be given by the clock, and the infant should be waked if asleep, since habit is an important factor in influencing the demands of the infant and its well-being. During the first three days, aside from the water it is given, a breast-fed infant receives sufficient nourishment from the colostrum, which it derives from the breast at intervals which are at first longer than those inaugurated after the milk begins to flow. With the infant which must be bottle fed from birth, the giving of a suitably dilute modification should be promptly begun, with an interval dependent upon circumstances of at first four and then soon of two hours, since the education of the stomach for this kind of food must be begun with care, and because at best the recovery of the normal loss of weight after birth and the establishment of a regular gain is slower with the bottle fed. The two-hour interval suitable for the first three weeks should, however, at the fourth week give way to a longer interval of two and a half hours, and this at the fourth month to three hours. This lengthening of the intervals is for two reasons: first, that the quantity and strength of the food are increased; second, that as the stomach takes on greater powers of digesting the food its secretions act upon the milk, forming compounds which remain longer in that organ.



Time should, therefore, be given for the stomach to empty itself before another feeding is given. This is usually longer with cows' milk than breast milk.

**Number of Feedings in Twenty-four Hours.**—These bear a relation both to the amount of food and to the length of the interval, and they decrease as the infant grows older. (See schedule, p. 142.)

**Night Feedings.**—During the first month, while the total number of feedings is ten and the interval is two hours in the daytime, two feedings may be given at longer intervals during the night hours from 9 P.M. to 7 A.M. When the number is reduced to nine at the beginning of the second month and the interval is made two and one-half hours, only one of these feedings should be given during the night. Feedings at night should be discontinued at the beginning of the fourth month, when the bottles are seven in number and the interval three hours. The first morning feeding may be at 6 or 7 A.M., and the last be given at 9 or 10 P.M. If the infant wakes during the night and is thirsty, plain boiled water may be offered. Elimination of the night feedings, by permitting undisturbed sleep and allowing the digestive organs a prolonged rest, is distinctly beneficial to the infant.

#### FEEDING THE NORMAL INFANT FROM BIRTH.

When necessity dictates that the infant must be artificially fed from birth, certain principles are now generally accepted which are applicable to the majority of normal infants. It is recognized that such an infant can assimilate a larger amount of fat than proteids, since the fat is absorbed with little change, but the proteids must be digested. Since this is an especially difficult function for the stomach to acquire when cows' milk replaces breast milk, the initial amount of proteids in the modification should not exceed 0.25 to 0.33 per cent. upon the first day, and this amount should be gradually increased, carrying with it a proportionate increase of the fat. The accepted ratio of fat to proteids for the first three to four months is three times as much fat as proteid (12 per cent. top milk, or upper third of bottle). From about the fourth month to toward the end of the first year, the proportion may then be fat double the proteids (8 per cent. top milk, upper half-bottle), and from that time on equal fat and proteids (plain milk). Such progression is best shown by the accompanying table from Holt. (See p. 145.)

Exact percentages, such as are shown in the table, can only be secured by prescription feeding with the aid of a milk laboratory, but approximate results which will serve the purpose in the average case may be obtained by the dilution of various strengths of top milk. Here, again, it should be stated that such schedules of percentages are intended only for the general instruction of the practitioner as to the amounts of fat and proteids which the average healthy infant *may* be able to take at these periods and those ratios which are more commonly successful; but these depend so largely upon the healthy digestion of the infant



and the care with which it has been fed at each stage that variations are very frequently necessary. Whatever criticism modified milk and percentage feeding have received in the past has arisen from the attempts of the physician to make the infant fit the formula rather than to find intelligently the proper formula for the infant. The needs of each infant must be studied by themselves, and increase or decrease of any of the elements of its food made after careful consideration of its digestion, stools, body weight, and general well-being. A schedule of percentages then serves simply as a guide which may be consulted to see how near we are approaching in the individual case to the averages which have been found advisable to secure normal well-balanced nutrition for an average infant. A large, vigorous infant and one that is small and delicate require different handling, but with such a schedule before us we shall be less liable to overfeed or, what is a still more serious error, to continue too long food which contains insufficient amounts of heat-producing and tissue-building elements.

SCHEDULE FOR AN AVERAGE HEALTHY INFANT, SHOWING PERCENTAGES OF FAT, SUGAR, AND PROTEIDS, AND QUANTITIES. (HOLT.)

Age.	Percentages of			Quantity for one feeding.		No. of feedings in 24 hours.	Interval by day.
	Fat.	Sugar.	Proteids.	Ounces.	Grams.		
Premature infants,	1.00	4.00	0.25	$\frac{1}{4}$ to $\frac{3}{4}$	7 to 22	12 to 18	1 to $1\frac{1}{2}$ hrs.
First to fourth day,	1.00	5.00	0.30	1 " $1\frac{1}{2}$	30 " 45	6 " 10	2 " 4 hours.
Fifth to seventh day,	1.50	5.00	0.50	1 " 2	30 " 60	10	2 "
Second week,	2.00	6.00	0.60	2 " $2\frac{1}{2}$	60 " 75	10	2 "
Third week,	2.50	6.00	0.80	2 " $3\frac{1}{2}$	60 " 110	10	2 "
Fourth to eighth week,	3.00	6.00	1.00	$2\frac{1}{2}$ " 4	75 " 125	9	$2\frac{1}{2}$ "
Ninth month,	3.00	6.00	1.25	3 " 5	90 " 155	8	$2\frac{1}{2}$ "
Fourth month,	3.50	7.00	1.50	$3\frac{1}{2}$ " $5\frac{1}{2}$	110 " 170	7	3 "
Fifth month,	3.50	7.00	1.75	4 " 6	125 " 185	7	3 "
Sixth to tenth month,	4.00	7.00	2.00	5 " 8	155 " 250	6	3 "
Eleventh month,	4.00	5.00	2.50	6 " 9	185 " 290	5	4 "
Twelfth month,	4.00	5.00	3.00	7 " 9	220 " 280	5	4 "
Thirteenth month,	4.00	4.50	3.50	7 " 10	220 " 310	5	4 "

**Feeding of the Average Normal Case from Birth.**—It is of the highest importance that an infant who is to be artificially fed should be started rightly, and that normal digestion be maintained during the critical period of the first three months. Started rightly the infant usually progresses favorably, while the digestion once upset is often very difficult to restore. For this reason, if we have good and sufficient grounds for believing that the mother cannot nurse her infant satisfactorily even for a short time, better results are attained by immediately beginning artificial feeding at birth, before the infant has lost

ground upon a hopeless milk. To secure for such an infant 10 feedings of 1 ounce each, to be given every two hours, and containing about 1 per cent. fat, 5 per cent. sugar, 0.33 per cent. proteids, we refer to the table, p. 141, and find that we must use 1 part of a "9-ounce top milk" and 11 parts of diluent. We would then remove with the 1-ounce Chapin dipper 9 ounces from the top of a quart bottle of good average milk upon which the cream has risen, mix them and use for the infant's food 1 ounce of this "9-ounce top milk," 1 ounce lime-water (10 per cent. of mixture),  $1\frac{1}{2}$  level tablespoonfuls of milk-sugar, and 10 ounces of boiled water. Put 1 ounce of this mixture in each of 10 nursing bottles, stopper with cotton, and pasteurize if deemed advisable. If dextrinized barley-water (see p. 138) is preferred, use 1 ounce of the "9-ounce top milk" with 11 ounces of this diluent and add the sugar. When we wish to increase the strength of the food in both fat and proteids, and to give larger quantities in each bottle, we continue to use the same "9-ounce top milk," and instead of making it one-twelfth of the mixture we make it one-tenth, one-eighth, one-seventh, etc., of the mixture, and prepare for the day any convenient quantity which is slightly in excess of our needs, and after placing the required amount in each bottle reject the surplus. If, for any reason, it is deemed advisable to change the proportion (3:1) of the fat to proteids, this is accomplished by choosing a different "top milk"—a stronger one (less ounces off top) to increase the fat proportion and a weaker one (more ounces off top) to decrease it. Whatever the total amount of food made for the day we use milk-sugar in the proportion of  $2\frac{1}{2}$  level tablespoonfuls for about every 20 ounces of food mixture; and if we employ lime-water, 2 ounces of it in every 20 ounces of food (10 per cent.) until good digestion is established, and then 1 ounce of lime-water to every 20 ounces of food (5 per cent.), both of these quantities of lime-water being counted in among the ounces of the diluent. Since the infant must first learn to digest cows' milk, beginning with percentages of the dissimilar proteids much lower than those in breast milk, we must neither expect the same stools nor the same prompt gain in weight as in a breast-fed infant. Our primary aim is not to give certain exact percentages, but to secure good digestion, which is best indicated by the comfort and sleep of the infant; and to push both the strength and quantity of the food forward as rapidly as the infant can take care of it, so that the infant who was started with fat 1 per cent., sugar 5 per cent., proteids 0.33 per cent. at birth shall be taking 3 to 3.50 per cent. fat, 6 per cent. sugar, and 1.50 per cent. proteids at the beginning of the fourth month, and fat 4 per cent., sugar 7 per cent., proteids 2 per cent. by the middle of the year. Since more than 4 per cent. fat is liable to disturb digestion, it will be thus seen that after the fat reaches 3.50 per cent. the proteids are pushed up more rapidly than the fat, being half the amount of the fat at five to six months, and approaching equality during the last few months of the year. Increases in quantity and strength should not be made with the suddenness apparently indicated by all schedules, but, when necessary, should be made gradu-

ally, covering several days. It is a safe plan when, with good digestion, the infant is not satisfied with the bottle, to first increase slightly the amount, and, if still unsatisfied, to increase the strength slightly every second day to the desired amount. The interval allows time to judge of the stools. One should not be too timid; slight disturbance or discomfort often occurs for a day or two with these changes. They indicate that we must stop the increase for the time being, until digestion has adapted itself to the new amounts, not necessarily a reduction of the food. Sharp or continued and increasing disturbance calls for a radical cutting down of the food, clearing out the bowel, and, with restored digestion, a gradual resumption. With artificial feeding, begun at birth, plain water plus lime-water is a satisfactory diluent in most cases. If cereal diluents are used they should not be strong (1 teaspoonful barley flour to 1 pint), and should be dextrinized. Unless cereals have already been begun they are a useful addition about the eighth month, and need not then necessarily be dextrinized, since it is better that the digestion should perform its own work if possible. For the same reason alkaline additions may be omitted at this time if practicable, as recent experiments in feeding young animals would seem to indicate that too long continuance of alkalies and antacids may have a deleterious influence upon development. This raises the question whether the tendency on the part of those who advocate alkaline diluents during the first year to postpone the giving of many articles of solid food until later periods than formerly, may not be due to the fact that under this system the normal development of gastric digestion has been delayed. Food subserves the two purposes of nourishing the body and furnishing fuel to maintain body heat. Any excess of heat formed must be given off, and this heat excretion is more difficult in summer. During hot weather less fat is required for heat production than in cold, and less can be assimilated. Fat percentages which are readily taken in winter often cloy in summer and cause loss of appetite or even actual disturbance, while the body will also be as well nourished with a smaller amount of food. Much subsequent difficulty will be avoided if, on the occurrence of fever or any acute illness, the food is at once diluted with one-third to one-half plain water, as digestion is always temporarily impaired.

#### **BEGINNING MODIFIED MILK LATER THAN AT BIRTH.**

In the case of all artificially fed infants who come under our supervision at any period later than at birth, it is advisable to investigate the feeding and to make an approximate calculation of the percentages which the child is receiving, in order to determine whether the various elements are being furnished in amounts adapted to its age and needs. This is especially necessary when the feeding has been carried on by the parents or friends. Such an investigation usually reveals the necessity for certain radical changes, and often for the recasting of much of the regimen if future trouble is to be avoided and proper



nutrition maintained. This may be true even in apparently well-nourished children, such as those who have been fed on condensed milk or, having done fairly well on proprietary foods, have been kept on them too long. The future of the infant demands a change, but the temptation is often to continue while the infant does fairly well. Good judgment is required in these cases. In the case of an infant who has just begun to gain and to show a better digestion after prolonged disturbance sudden changes should not be lightly made, but the necessary elements of milk should be introduced or gradually increased to replace the others which are decreased and withdrawn. Young infants doing well upon condensed milk may be carried on for a time by the addition of gravity cream (16 per cent.) in a quantity equal to that of the condensed milk in each feeding. This may then be replaced by a low formula in which the fat is double the proteid. If the change is to be carried out at once, since most children who have been fed condensed milk have received mixtures of uncertain strength, owing to the difficulty in estimating the actual bulk of the milk added to the water, and since at best these contain usually low fat and proteid percentages with a relatively high sugar, it is here even more than ever necessary to follow the fundamental rule when beginning to feed modifications of cows' milk to any infant and to commence invariably with low formulæ, working up to higher ones. This rule holds good in all cases, whether it be in giving supplementary feedings to an infant at the breast, or in weaning a nursed infant, or beginning the feeding of cows' milk either at birth or at any subsequent period of infancy. It is the first commandment of infant feeding and the second is: do not continue with low formulæ, but increase the fat and proteids as quickly as digestion will allow.

#### ATTENTION TO DETAIL IN INFANT FEEDING.

Thoughtful attention to detail is a prerequisite of success in almost every process and business. The overlooking or slurring of a single matter may vitiate the whole result. Eventual mastery of the situation is often reached only by the most painstaking inquiry, investigation and observation and by insistence that no possible contributory factor should go unrighted. Time must be taken to secure the past history of the infant, to write out clearly the directions to be followed, and to catechize mother and nurse as to the practical application of such directions. The clearest instructions are often neglected or misunderstood. When methods have been once put into use it is best to insist at a subsequent visit upon a detailed account of each step in the attendant's own words, as important errors are often only brought to light by this means. Actual inspection of the materials and processes may be necessary to detect some serious fault. Observation of the infant and its management frequently reveals much which requires change. The symptoms which most deeply impress the attendant are often the least important,

and the really suggestive ones must frequently be extracted by cross-questioning.

**Important Adjuncts to Digestion.**—There are other matters besides the preparation and proportions of the food and the quantity, interval, and number of the feedings which, when thrown into the right or wrong side of the balance, assist or defeat our purpose. These will be referred to briefly in this section, as they are elaborated in other parts of the book. After each feeding every infant's mouth should be washed with boiled water or boric acid solution. The functions of the skin should be maintained by the daily bath. Normal crying, which develops the lungs and thus favors oxygenation and muscular action, should be sought rather than repressed. After feeding, the infant should be laid down. It should not be picked up because it cries, but a change of position often makes it comfortable. Walking, patting, rocking, and bouncing an infant are to be denounced. They do not relieve pain, but further tire out the nervous system. Abundant sleep at regular hours should be encouraged. Amusements and numerous or complicated toys should not be forced upon the infant's attention. With reasonably free play for the exercise of legs and arms, the infant should be allowed to lead a vegetative existence. The every-day marvels of its environment and an occasional simple toy of the plainest kind furnish all the stimuli which are advisable. Undue stimulation of the nervous centres is usually at the direct expense of the organs of digestion.

**Oxygen in Fresh Air as a Food.**—In discussing the composition of food no thought is usually given to that most important element which enters the body not by the mouth, but by the lungs. A large proportion of the nutrition and tissue change, together with the production of heat and energy, is dependent upon the combination of the other elements with oxygen, of which the supply must be continuous. The daily feedings of other food rarely exceed ten; so great, however, is the demand for oxygen that this must be supplied to the infant from twenty-five to thirty-five times each minute. This form of statement will serve to emphasize the immense importance of fresh air, which must be secured, not only by more than ordinary attention to the ventilation of the apartment, but by taking the infant into the open air as early and as much as the season and the weather allow. In inclement weather, at least once a day, the infant should be dressed as for going out-of-doors and all the windows of the room thrown wide open. By attention to this one factor, failure is often turned into success.

#### COMMON COMPLICATIONS IN INFANT FEEDING.

**Vomiting.**—This may be acute or more or less persistent.

**Acute Vomiting.**—When not an initial symptom of some acute disease, vomiting is either due to the food itself or to some factor which temporarily arrests or disturbs the process of digestion. These are not always distinguishable. When an infant otherwise apparently well vomits its



food the rule is to omit the next bottle entirely or give in its place plain water and to dilute the following one one-half with boiled water. If the vomiting is repeated or there have been from the first other symptoms of disturbance it is wise to sweep any remains of undigested food from the stomach and bowels with minute doses of calomel (0.0005 gm. [ $\frac{1}{16}$  gr.] for five to ten doses) and to give barley-water, egg-water, whey, or plain water for twelve or more hours until the stomach regains its tone and appetite returns, beginning, then, with temporary low dilutions of the usual food. When no other cause can be discovered in hot weather it is often a safe rule to reject the remaining supply of food which has been made up and to await the arrival of a fresh supply of milk on the morrow.

**Habitual Vomiting.**—More or less habitual rejection of larger or smaller quantities at varying intervals after the food has been taken calls for careful investigation, both of the food and of the plan of feeding. Babies whose food comes up easily should be laid down at once after feeding with as little movement as possible and care taken that the abdominal bands are not too tight. Accurate observation of the time, amount, and appearance or odor of the vomitus should be insisted upon. We distinguish for practical purposes between the vomiting of the larger part or the whole of the food ingested at a meal and the spitting up of a teaspoonful or two; and also whether these occur within a few minutes after the food is taken or some time later. Spitting up of small quantities may occur with eructations of gas or, as in healthy breast-fed infants, be due to the rejection of an excess of food from an overfilled stomach when the peristalsis of digestion begins. This latter form occurs soon after the meal is finished and the food is but slightly changed. It is best met by a reduction in the quantity given. To be differentiated from this is the spitting up or vomiting of sour-smelling fluid or curds which takes place after digestion is under way. This may be at first of smaller or larger quantities, but there is a tendency for the amount to increase and to contain mucus from the stomach. This is very commonly due to too much fat in the food and is best treated by a sharp reduction in the fat percentage. In other cases too much sugar may be productive of the same difficulty and this must be lessened in amount. Spitting up and vomiting should always receive immediate attention and not be allowed to continue, for not only has the habit, when once established, a tendency to continue or return upon slight provocation, but the underlying causes soon lead to disturbances of the gastric mucosa, as evidenced by the increased secretion of mucus. Having corrected any discoverable faults, one or two teaspoonfuls of lime-water given shortly before each feeding is a helpful measure. This is rather more effective than increasing the amount of lime-water in the food, although where it already enters into its composition it may be raised temporarily from 5 per cent. to 10 per cent.

**Habitual Constipation.** An infant's bowels should move at least once daily. When this is not the case, the infant is liable to be uncomfortable, restless, and to sleep badly, even if it does not present worse

symptoms of colic and flatulence, and measures should be taken for its relief. Constipation is the rule with infants fed upon condensed milk which is seriously deficient in fat, and it may occur in those given modifications of cows' milk containing low percentages, and is overcome by increasing the percentages of fat if they are below the usual schedule averages. Care should be exercised, however, not to go to the other extreme and exceed the amount of fat which the infant can care for without disturbance. But few infants can take more than 4 per cent. fat without trouble. It has only recently been recognized that there is also a form of constipation with dry, hard stools due to excessive fat in the food. If the milk has been sterilized, changing to pasteurized milk, or, better, if a pure fresh article can be secured, to an unheated milk, may solve the difficulty. Proteids in excess, especially with low fat, may give rise to hard, dry feces, which, when broken, show a granular surface with small white particles, although it is perhaps more common for too much proteid to cause loose, undigested, curdy stools. Decrease of proteids and moderate increase of fat will remedy this condition. On the whole, however, relief of constipation is most commonly achieved by a judicious increase of both fat and proteids, which increase the unconsumed residue. Constipation should be regarded as a condition or a symptom rather than a disease, and is due most commonly to diet, lack of proper training, or to muscular inefficiency. Dietetic measures should always be given a fair trial. Even young infants can be trained to have their stools at regular times. Gluten suppositories or an occasional small and simple enema are far preferable to the habitual use of laxatives.

**Colic.**—This term is often applied loosely by parents to any condition which causes the infant to cry and draw its feet up toward the abdomen. It may arise from insufficient protection of the abdomen and extremities from cold, but is chiefly caused by indigestion due to excessive proteids and is accompanied by flatulence. It is relieved by heat to the abdomen and extremities and stimulant aromatics like dilute warm peppermint-water; a small enema is frequently effective. These tend to further disturb digestion, if given frequently. The exciting cause is to be removed and the condition cured by the reduction of the amount of proteid (casein) in the food, or by measures which increase its digestibility.

#### STOOLS.

The first stools of the newborn infant consist of dark-green meconium, the accumulated secretions of the intestinal tract. As the flow from the mother's breasts becomes established, the meconium passed in the first days is replaced by bright, orange-yellow feces. If the milk is scanty this may be divided into small, yellow masses or flakes, surrounded by green mucus. These small, fatty masses are often mis-called curds and much misunderstanding thereby arises. True, hard curds are not formed from breast milk. In all questions of insufficiency

of breast milk and where disturbed stools are reported in nursing or bottle-fed infants the physician should insist upon having the napkins kept for his inspection and learn to distinguish the different types and their significance. Thus we distinguish the lighter green stools of indigestion from the small and frequent dark-green, mucoid stools which show little or no milk residue and indicate reduced intake from whatever cause. If the residue of breast milk be bright yellow, it is not a question of indigestion, and with increased ingestion of breast milk the green biliverdin coloring the mucus will, when mixed with more residue, appear in the form of the normal, yellow bilirubin of the bile. The absence of residue in the stools of bottle-fed infants should always lead to an investigation of the amounts which they are actually taking or retaining, although the stools are not as typical as in the nursing infant. What has been said of this characteristic type of stools should not be misapplied to other types of green stools.

The normal stools of infants receiving cows' milk are yellow, but have not the orange tint of the breast fed. When other substances are added to the diet the color is often influenced. Certain infant foods, especially when given unmixed with milk, give their own characteristic stools. Imperial Granum, Malted Milk, and foods of the latter class give dark or brownish stools. Barley-water, given alone, produces a somewhat slimy stool, often mistaken for mucus, especially when the movement shows mucilaginous particles.

The disturbed stools of artificially fed infants appear in very varied and frequently mixed forms, but certain of the types may be mentioned.

In normal stools it should be possible to spread the fecal matter out by pressure with the napkin, as a smooth, homogeneous, buttery mass. Too much proteid may either produce constipation or diarrhea. If constipation, the stools, especially when the fat is low, are friable, and when broken appear to be made up of small, whitish granules. Loose stools, in which the milk residue is whitish and in small flakes or masses, are usually denominated curdy, even when these smooth out readily and are soluble in ether, showing them to be mainly fatty masses, although the condition is caused usually by proteid indigestion.

True curds are firmer, and the most typical are rounded, tough masses, yellow on the surface and white within, somewhat resembling grains of Indian corn. These are formed in the stomach, where the coagulation of the milk takes place, by the shrinking of considerable masses of the denser products of paracasein with acids, which the stomach fails to digest, and are passed on into the intestine, where they cannot be disintegrated and act as disturbing foreign bodies. They presuppose a relative excess of acidity in the stomach, and may be brought about by (a) the presence of lactic or other acids formed in, but not secreted by, the stomach; (b) lactic acid in the milk which has been allowed to become slightly sour either before or after modification; (c) hypersecretion of hydrochloric acid, the total acids present forming



under favoring circumstances more large and tough curds than the stomach can digest. These formations can be prevented by peptonization, or by the addition of alkalies which neutralize any acid in the milk, or acids which may be present in the stomach when the milk is ingested, and also delay or prevent the action of rennet and of the hydrochloric acid subsequently secreted. Other contributory measures are the use of a fresh milk kept properly cooled; pasteurization, which kills lactic-acid-forming bacteria; further dilution of the milk; and the use of gruels as mechanical attendants of the curd.

**Green Stools.**—No very satisfactory explanation of the green stool has been advanced. The color is due to the changing of bilirubin to biliverdin in the intestine under disturbed conditions. A sharp distinction in prognostic value should be made between a fairly well-digested yellow movement, which turns green on the surface quickly on exposure to the air, even before the diaper is removed, and the all green or green and white stools. The former (yellow turning green) are much more favorable. Stools produced by calomel are often green, this color disappearing as soon as the action of the drug is at an end. Green stools often occur in epidemic or endemic form in wards of institutions caring for bottle-fed infants, and they are probably due to infection by special bacteria. The same probably is true of the green stools of summer diarrhea which are of protean form.

**Fatty Stools.**—Excessive fat in the food may cause loose stools, which may even have a greasy appearance, or large, hard, dry stools. More frequently they are sour-smelling, yellow, greenish-yellow, or even green stools, having the curdled appearance of scrambled egg, and may, if continued, contain mucus. Excess of sugar may at times be responsible for a similar condition. In certain cases, where the intestinal digestion and absorption are at fault, large, gray, putty-colored movements are passed, with a peculiar odor, which is often ammoniacal. They contain an excess of fat, which should be reduced in the food, and small doses of sodium phosphate (0.325 to 0.650 gm. [gr. 5 to 10] t. i. d.) given to re-establish proper hepatic secretion. In older children, upon a somewhat mixed diet, especially in those showing the large abdomen and other signs of rachitis, the odor of the stools is often very foul, filling the room. Restriction of the diet to milk alone, and the use of some intestinal disinfectant such as salol, gr. 1, four times a day, will often prove effective.

**Mucous Stools.**—Mucus, to some extent, is an integral part of all fecal movements, but, when thoroughly incorporated, does not appear as such. It appears in excess in most conditions of prolonged irritation, whether from the presence of hard, fecal masses or from faulty digestion of the intestinal contents.

**Watery Stools.**—Thin, loose stools of a yellow color are seen in the diarrheas of summer. Profuse, watery stools containing only occasional flakes of mucus, the so-called rice-water stools, characterize the intense form of intestinal poisoning by toxins of bacterial origin, which constitutes a true cholera infantum.

**Bloody Stools.**—Blood may appear not infrequently, and varies considerably in its import. A hard, constipated movement may be streaked with bright blood from a small tear of the anal mucous membrane. Such streaking in nursing babies without temperature does not often recur after a moderate dose of castor oil. In severer intestinal conditions—colitis, ileocolitis, and certain epidemics due to bacteria of the Shiga group—blood is perhaps even more liable to appear in the stools as the result of intense congestion of the mucosa than of actual ulceration. Hemorrhoids are seldom seen in infancy. Small, bleeding polyps are of occasional occurrence. It should never be forgotten that small, frequent stools of blood and mucus only, passed with straining, point strongly to intussusception.

#### FEEDING IN DIFFICULT CASES.

While some children will thrive upon almost any kind of feeding, and the majority of the others upon carefully adjusted formulæ of modified cows' milk, there still remains a mixed class which try our knowledge and ingenuity to the utmost. A few, very few, indeed, are disturbed by small quantities of cows' milk, or seem to be unable to digest enough to enable them to secure proper nutrition, and these are better wet-nursed if practicable. But this view of the case should not be assumed lightly or without intelligent trial, since the vast majority of those who come into our hands in this apparent condition are the wrecks left by ignorant, injudicious, and unscientific attempts at feeding, which have so deranged their digestions that formulæ suited to a normal infant of that age are not tolerated. This is the class which has caused most of the dissatisfaction with percentage feeding, because the stock formulæ fail in the majority of instances when applied to these difficult cases. The more different kinds of infant foods which have been tried in rapid succession, and the greater the actual or relative loss of weight the infant has sustained, the more difficult the problem. If the practitioner will always start upon the principle that no infant whose percentage feeding is begun at any time later than birth will be liable to digest the formulæ laid down for the age at which such feeding is begun, but that he must start with a weak formula which can be brought up more or less rapidly to stronger ones, the most frequent source of failure will be avoided. Furthermore, practically all infants who have been variously fed before coming into our hands had best be considered as having disturbed digestions, which must be restored by beginning with easily digested mixtures before higher ones can be attained to. A full history should be secured, in such cases, of the previous attempts at feeding, the proportions of the ingredients, the quantities, the daily number of bottles, the intervals employed, and of the behavior of the infant with the various foods; especially with reference to sleep, and to the occurrence of vomiting, colic, and the appearance and frequency of the stools. By such means only are we in a position to discover the underlying errors, and to avoid



continuing or duplicating them. Many infants are declared to be unable to digest cows' milk when the difficulty has depended upon too high fat, or too high proteid, or too much of both. In reality, suitable modifications of cows' milk give the best results in the vast majority of cases. Our first care must be to restore digestion. To that end the quantity of each feeding and the interval between feedings must be carefully regulated. If the infant has been given the bottle irregularly or every two hours, the interval should be increased to at least two and one-half hours; or better, every three hours, especially if the child has passed the age of three or four months. Frequent night feedings allow no period of necessary rest to the digestive organs.

When the disturbance is recent or the development fair the quantity given may be determined by the age (see p. 142), but infants who are considerably under weight are in danger of being overfed in amount. One often sees infants of five, six, or more months, who still weigh only about seven or eight pounds, scarcely more than their birth weight. These should neither receive the quantity laid down for normal infants of their months, nor the smaller amount which a normal infant of their weight would be given during the first few weeks of life. The stomach has grown somewhat in its normal capacity, but has either suffered dilatation from overfeeding or is confronted with that danger. Overfilling will often defeat the expectations from careful modification of the food. A mean must be struck between the age and weight, and experience teaches us that this is, for such an infant, from three to four ounces. Such infants are often ravenous, having secured little nourishment from the food which has been ingested, but has not been assimilated. When these infants are placed upon low formulæ and reduced quantities their apparent hunger may continue, but may be disregarded until indications appear showing that better digestion is established. Where we have reason to believe that the previous difficulties with the digestion of milk have arisen from too strong formulæ or when beginning milk feeding for the first time, we should always begin with a low formula or a mixture which we are reasonably sure is within the digestive power of the infant. These infants rarely do well at first with a fat percentage more than twice that of the proteids, such as those made from the top half of the bottle, 15 ounces, or an 8 per cent. top milk: fat, 1.00; sugar, 6.00; proteids, 0.50; or, fat, 1.50; sugar, 6.00; proteids, 0.75. Often similar dilutions of a 6 per cent. top milk (20 ounces from bottle) are better to begin with: fat, 0.75; sugar, 6.00; proteids, 0.50; or fat, 1.00; sugar, 6.00; proteids, 0.66, etc. While with marasmic infants whose powers of absorbing fat are notably poor and in vomiting cases dilutions of plain milk at first succeed the best: fat, 0.50; sugar, 6.00; proteids, 0.50; fat, 0.75; sugar, 6.00; proteids, 0.75, etc. These cases have less trouble with the sugar than with the other elements, and it should never be omitted in these formulæ. Either alkaline diluents or plain or dextrinized barley-water may be used. But the latter is perhaps more commonly successful, although if one plan fails another may give good results. The parents should be made to understand at the outset that

our first aim is to establish normal digestion of low formulæ, as shown by the disappearance of vomiting and colic and by the return of the stools to a more normal color and consistency. To this end a regular inspection of the stools is even more important than seeing the infant itself. The securing of an immediate gain in weight does not compare in importance with this improvement of the symptoms and stools; and, although systematic weighing should be carried on for the guidance of the physician, too much anxiety should not be felt over a stationary weight or even the further loss of a few ounces during the first few days of the adjustment period, provided the symptoms of digestion are improving with less restlessness, less flatulence, more gastric tolerance, and better stools. These low proteid percentages should not be too long continued. It is as serious an error to persist too long in the use of low formulæ as it is to begin with those that are too high. Just as soon as improvement in the symptoms is manifest the strength of the food — *i. e.*, the amount of milk or top milk in the formula—should be pushed gradually forward and in due time a gain in weight will be inaugurated, remembering, however, that it is a golden rule, when an infant is gaining satisfactorily, to let well enough alone and to postpone changes until they are clearly indicated. It requires careful judgment at times to determine whether an infant requires more in quantity of the same formula or the same quantity of a higher formula. The stools may here be our best indication. If despite care we overstep the limits of digestion, or there is disturbance from any other cause, we should not hesitate to sharply cut down the strength of the food, which may be again gradually restored as conditions improve. The mistake should not be made, on the other hand, of changing the food too often, for slight disturbances do not always indicate a change, since the cause of the difficulty may be found upon investigation to be due to carelessness in the preparation of the food, to dirty or cold bottles, or to disregard of instructions concerning feeding. The successful management of difficult cases comes partly from experience, but is also dependent largely upon the amount of intelligent study and careful supervision of the case. After investigating the previous management of the infant, we should endeavor to form a definite plan of action, based upon the information which we have secured and the apparent indications, and this plan should be fully tested before adopting another. The plan which we adopt may be one that may have been tried before, stripped of its gross and palpable errors, or it may be an entirely different one; but everything which we do should have a distinct reason and purpose; not with the forlorn hope that anything which is different may hit the mark. If any one plan has been previously tried for some time a radical change may be crowned with success. This is especially true in vomiting cases.

It is a good rule, upon assuming the care of a new case, and always when beginning a new line of feeding, to start afresh by clearing out the bowels with calomel or castor oil. Sucking nipples, "pacifiers," or "comforts" are frequent appurtenances of these cases, and should be unqualifiedly condemned and their use stopped. They overtax the

salivary glands, favor the continuance of vomiting, are unhygienic, and introduce dirt and bacteria into the alimentary tract besides inaugurating a train of most undesirable habits. Such infants must be under close observation for a time. The mother should understand that a single food prescription will not dispose of the matter. Not only will some changes and variations probably be necessary before the combination is found upon which the infant does best; but once found this cannot be continued indefinitely, but must be altered from time to time as improved digestion and assimilation allow us to do so. Many of these cases fall into two groups in which the symptoms are either chiefly gastric or chiefly intestinal. The former have as their prominent symptoms vomiting or habitual spitting up of their food; the latter, colic, eructations of gas, furred tongue, flatulent distention of the abdomen, constipation, or frequent loose stools which may contain mucus.

**Vomiting Cases.**—Vomiting which has become habitual often proves the most difficult obstacle to overcome, as it prevents the retention of a sufficient quantity of food. If the disturbance has been very marked, it may be wise to stop all other food and rest the digestive organs by giving only dextrinized barley-water for one or two days, and then begin by adding a teaspoonful of milk to each bottle, promptly increasing the amount added as it is tolerated. Daily lavage of the stomach for a few days may be necessary at the outset, especially where there is much mucus. In some of the worst cases, having washed the stomach once each day, the food must be given at all the feedings by the stomach tube and funnel (gavage) and will only be retained when so administered. It is important to pinch the tube when it is removed and withdraw it quickly so that no food will be deposited in the pharynx to invoke gagging and vomiting. Whey is often well borne when other food is omitted, possibly because of its low fat; but its use alone should not long be continued. Excess of fat and sugar must be avoided, but the necessity for retaining these, especially the sugar, to some extent must not be forgotten or the proteids will be called upon to furnish body heat. Many of the artificial foods, which contain much sugar (soluble carbohydrates), we have come to recognize as common exciters of vomiting and loose stools and are to be avoided in vomiting cases. Firm, cheesy clumps in the vomited matter call for increased alkalinity or reduction of the proteids or also, perhaps, for peptonization of the food. Dilutions of plain milk are the best to begin with. The interval should not be less than three hours for young infants, and if we keep the fat low smaller quantities of a more concentrated food are more liable to be retained than larger ones.

**Intestinal Cases.**—These, as stated above, are usually distinguished by the occurrence of colic and tympanites, with constipation or curdy, loose movements. These types almost always arise primarily from difficulty with the digestion of the proteids or more specifically of the casein of cows' milk, which calls for reduction in its amount or measures which will add to its digestibility. Ordinarily relief is best effected by further dilution of the milk, allowing the stomach to perform more



thoroughly its part in digestion and so lessening the burden falling upon the intestine. To be effective, as in all changes in food to meet special symptoms, the reduction should be a radical one, with subsequent gradual increase. These children usually digest their proteids better in the presence of a fat percentage twice to three times that of the proteids, which are about the normal proportions in breast milk. Lowering the proteids in this class does away with the colic and flatulence in constipated cases and the indigestion which leads to diarrhea. Increasing the fat relieves constipation in the former group, the residue of unabsorbed fat making the stools softer. Care should be exercised, however, not to overdo this, since excessive fat will disturb the stomach and cause regurgitation or vomiting, or may be too laxative and cause loose movements. Few if any infants can exceed 3 per cent. fat during the first three to four months, or 4 per cent. fat during the remainder of the first year, without trouble ensuing sooner or later.

#### **OTHER MEANS OF INCREASING THE DIGESTIBILITY AND ABSORPTION OF PROTEIDS.**

If a fair trial of dilution and gradual increase of proteids fail to relieve the symptoms of proteid indigestion, or their gradual increase to a point necessary for nutrition be followed by a return of the difficulty, other measures may be adopted. The most recent is that of "split proteids," or the employment of whey as part of the diluent of a rich top milk, so that with a suitable amount of fat there shall be but a small amount of casein, the remainder of the proteid required being made up of the soluble proteids of whey. The use of dextrinized barley-water, which furnishes some absorbable vegetable proteid, may prove especially successful in the intestinal type of cases. Peptonization may also serve us, although less effective in the intestinal than the gastric class. The influence of alkalies, lime-water, and bicarbonate of soda, in preventing the formation by the gastric secretions of denser and less easily digested compounds, should also be kept in mind. Although not ordinarily recommended for the food of infants, condensed milk, begun with the proportion of one level teaspoonful to four ounces of water, will frequently be successful when other efforts fail. It should, of course, be increased, and in due time changed to modified milk. Favorable results are more readily attained in private practice, even among the poor, if reasonable co-operation can be secured from the parents, than in large institutions where, especially when crowded and the air space per infant less than 1000 to 1200 cubic feet, infants under six months of age show a large mortality. One important factor, at least in institutions, is probably "crowd poisoning," from which authorities on military matters state that even soldiers on the march are liable to suffer, although in the open air, if allowed to retain close formations. Some of the worst cases of malnutrition will not thrive on usual modifications of milk, and show continuous loss of weight and require special

measures to stay the loss and re-establish a gain. To these, especially if there be vomiting, whey may be given for a few days, but it must not be continued too long alone. Better still is whey with the white of one fresh egg to each 10 to 16 ounces, strained and salted. Important adjuncts in the treatment of these especially difficult cases are liquid peptonoids, 5ss-j, with each feeding, which serves more to stimulate absorption than to furnish nourishment. Difficulty in proteid digestion of even small amounts of casein is often assisted by elixir lactopeptine, 15 to 20 drops in each bottle. A temporary respite from loss of weight and a slight gain which may be continued by careful feeding are sometimes effected by protonuclein, 0.06-0.12 gm. (gr. 1-2), four times a day. Once started upward, these malnutrites must be most carefully guarded from upsets. Partial breast feeding may be their only salvation in institutions, even if but a few daily nursings can be secured. Normal development demands proper amounts of fat, sugar, and proteids; but children whose digestions have been long disturbed vary greatly in the amounts of each which they can properly care for; so that while our aim is to get the elements back to somewhere near usual proportions, when it can be safely done, the food of many of these children must differ considerably from that of the average child. With digestion restored and a proper amount of food for their individual requirements, gain in weight, although often long delayed, will surely come.

#### PEPTONIZED (PANCREATIZED) MILK.

Peptonization has two purposes: to increase the digestibility of milk, and to increase the amount of its casein which the child can take without disturbance. The usual peptonizing tubes contain 5 grains extractum pancreatis and 15 grains sodium bicarbonate. The process, therefore, is analogous to intestinal digestion in an alkaline medium. It is chiefly useful where the difficulty is gastric, as it predigests more or less of the casein, transforming it into non-coagulable albumoses and peptones. But whether more or less of the casein is transformed, the alkalinity of the bicarbonate of soda, especially if it has been heated, restrains the action of the rennet and acid of the stomach, preventing to any extent the formation with the remainder of tough acid paracasein curds. In short, gastric digestion is more or less cut out. With vomiting cases the fat should be kept low.

It is also useful, though less frequently, where the trouble is intestinal. The degree to which the predigestion of the casein is carried depends upon the length of time during which the action of the ferment is allowed to continue. Milk may then be partially or completely peptonized. For young infants the time should be rarely less than twenty to thirty minutes to be effective. Heating the milk to the boiling point kills the ferment and stops further action. Complete peptonization of all the casein requires about two hours and is at times necessary. All the feedings for the day may be peptonized in bulk, or, what is often better, a small portion of powder may be added to the warmed bottle a definite



time before each feeding. The contents of each peptonizing tube is sufficient to transform 1 pint (16 ounces) of milk. In peptonizing the total food prepared for the day, if the mixture contains 8 ounces of milk or top milk, use one-half tube, etc. In adding to the bottle before feeding, use a little more than one-sixteenth of the tube for every ounce of whole milk or top milk which entered into the preparation of the bottle. Following this plan the nurse may be directed to divide the contents of a peptonizing tube into a specified number of powders. Peptonizing should not be continued indefinitely, else the functions of the stomach become weakened by disuse. As soon as possible it should be stopped by reducing the time five to ten minutes each day until withdrawn. Peptonization is probably employed less frequently than formerly, as it is often disappointing in its results except in selected cases, and it in nowise removes the necessity for intelligent modifications. Continued for more than fifteen minutes it develops a slightly bitter taste, which is not, however, usually objected to by infants.

Peptogenic milk-powder is composed of pancreatin, sodium bicarbonate, and milk-sugar. One measure or capful is required for each pint of milk. If employed for young infants or those with difficult digestion, the usual mixtures advised are too strong and the powder should be added to formulæ suited for the case. As pepsin always contains rennet, it will, if added to milk, curdle it.

#### WHEY AND CREAM AND WHEY MIXTURES.

Many infants experience so much difficulty in digesting the casein of cows' milk in sufficient quantity to maintain nutrition and to provide for increase of weight, that the attempt has been recently renewed to increase the amount of easily absorbable proteid in the food by the use of whey which contains the soluble proteids of the milk. This plan, which is especially capable of variation in laboratory feeding, has been called that of "split proteids," although such admixture of soluble proteids and casein exists in all milks and in all modifications of milk. It really consists in increasing the amount of the soluble proteids in an infant's food without increasing in the usual proportions the amount of casein. This may be brought about by using, as the basis of the food, whey, in which the soluble proteids of the milk have been largely separated from the casein by clotting the latter with rennet. To reduce this to a scientific basis it is necessary that we should start with a clear conception of the composition of whey. The following table from Van Slyke gives actual analyses of whey made from poor, medium, and rich milks.

	WHEY.		
	From poor milk containing 3 per cent. fat	From medium milk containing 4 per cent. fat.	From rich milk containing 5 per cent. fat.
Total solids . . . . .	6.87	6.96	7.38
Fat . . . . .	0.28	0.30	0.30
Total proteids . . . . .	0.69	0.87	1.08
Sugar and ash . . . . .	5.90	5.79	6.04
Water . . . . .	93.13	93.04	92.62

From this we may deduce that whey, such as is made in cheese factories, if prepared from a good average 4 per cent. fat milk, will contain about 0.30 per cent. fat, 0.90 per cent. proteids, 5 per cent. sugar, and 0.75 per cent. mineral salts. Wheys prepared for home modification show considerable variation in their constituent percentages owing to the different milks used, the different preparations of rennet, and the method employed in making the whey. Ordinary methods give a very cloudy whey. This cloudiness is due to finely divided particles of the junket clot (paracasein) and more or less fat. Whey made from whole milk will contain more fat than that made from a milk from which the cream has been largely removed. The following method is recommended for securing a fairly fat-free whey or for making cream and whey mixtures:

**Method of Making Cream and Whey Mixtures.**—Secure a quart bottle of good average milk upon which the cream has risen. Remove with the Chapin dipper the upper 5 ounces of the cream layer, which, when mixed, will contain about 20 per cent. of fat, and preserve this for further use. Pour the remainder of the bottle (about 27 ounces) into a double boiler, the lower portion of which contains tepid water, and add one tablespoonful Shinn's liquid rennet, or one Hansen's junket tablet, or one tablespoonful of Wyeth's liquid rennet. Mix thoroughly. Place a chemical thermometer in the whey, and heat slowly up to 155° F. (68° C.) to destroy the rennet ferment, which otherwise would clot the casein of the cream or top milk when subsequently added to the whey. Heated beyond 155° F. the albumin, part of the soluble proteids, will be coagulated and the nutritive value of the whey reduced. As soon as a solid curd forms cut this crosswise into small pieces with a table knife to facilitate the escape of the whey, and while continuing to heat to 155° F. use the flat of the knife blade to assemble and press together the pieces of curd. This increases materially the yield of whey, and the curd finally contracts with heat and manipulation into a rubbery lump the size of the palm of the hand. Straining through a wire strainer now gives 20 ounces or more of moderately opaque yellowish whey, upon which but little fat rises on standing. Adding to 20 ounces of this whey varying amounts of the top 5 ounces of cream (20 per cent. fat), previously removed, will give us a series of formulae suitable for most purposes where cream and whey mixtures are required. By removing and using the top 6 ounces (17 per cent. fat) or top 7 ounces (15 per cent. fat), mixtures may be obtained with a lower fat percentage; or by using more of these top milks in the mixture the same amount of fat with a larger proportion of casein in the proteids.

WHEY AND CREAM MIXTURES, MADE FROM 20 PER CENT. CREAM (TOP FIVE OUNCES OF ONE QUART BOTTLE) AND TWENTY OUNCES OF WHEY FROM REMAINDER OF BOTTLE.

		Fat percentage.	Sugar percentage.	Proteid percentage.
20 oz. whey + 1 oz. cream (20 per cent. fat)	"	1.00	5.00	0.90
20 " " + 1½ " " " " "	"	1.50	5.00	1.00
20 " " + 2 " " " " "	"	2.00	5.00	1.10
20 " " + 2½ " " " " "	"	2.40	5.00	1.15
20 " " + 3 " " " " "	"	2.75	5.00	1.20
20 " " + 3½ " " " " "	"	3.15	5.00	1.25
20 " " + 4 " " " " "	"	3.50	5.00	1.30

Bartley suggests the addition of the white of a fresh egg and also of a full tablespoonful of milk-sugar dissolved in the whey of each quart bottle of milk.

As soon as digestion is re-established upon a whey diet a cautious attempt should be made to add to the cream and whey mixture plain milk or a larger bulk of top milk with a lower fat content, in order that the stomach may again resume its function of digesting casein, which alone supplies the form of proteid required for well-rounded development. Whey feeding at best should be but a temporary expedient, unless it is combined not only with extra fat, but also with increasing quantities of casein beyond those contained in the formulæ of the above table. Although invaluable for short periods in certain emergencies, the continued use of whey alone for long periods is disastrous if not indirectly fatal to the infant.

#### CONDENSED MILK.

This is cows' milk from which a large part of the water has been removed by evaporation in vacuum pans, in which boiling takes place at a lower temperature than under ordinary conditions. As a part of the process it is also sterilized. It may then be sold in bulk for immediate use, or sealed in cans with the addition of sugar. More commonly cane-sugar is added as a preservative in proportion of about six ounces to the pint of condensed milk. Many of the so-called evaporated creams are no richer in fat than average condensed milk. There is no uniformity between the various brands of condensed milk found in the market. Illinois, New York, Ohio, and Oregon alone have laws regulating the quality. Elsewhere brands are sold which analyses show to be evidently made from skimmed milk. The following table, founded upon an analysis of "Eagle Brand," is fairly typical, furnished to Chapin by the United States Department of Agriculture:

Cows' milk for comparison per cent.		Condensed milk per cent.	With 15	With 13	With 11	With 9	With 7
			parts water 1 to 16 per cent.	parts water 1 to 14 per cent.	parts water 1 to 12 per cent.	parts water 1 to 10 per cent.	parts water 1 to 8 per cent.
4.00	Fat . . .	8.44	0.53	0.60	0.70	0.94	1.05
3.50	Proteid . . .	7.23	0.45	0.52	0.60	0.72	0.90
5.00	Sugar { cane, 41.52 milk, 11.69 }	53.21	8.93	8.80	4.43	5.32	6.65
0.70	Salts . . .	1.90	0.11	0.13	0.15	0.18	0.22
86.80	Water . . .	28.41	95.08	91.95	94.12	92.94	91.18

Comparing this with average percentages of fresh cows' milk, it will be seen that Leeds' estimate that one part of condensed milk equals about two and one-half parts of fresh milk is approximately correct. It will also be evident that when diluted with 12 to 16 parts of water, according to the usual directions, both fat and proteids are very low. But these directions are only exceptionally followed by the laity, who more commonly put a part or the whole of a teaspoonful of condensed

milk in a "cupful" of water or even in the usual 8-ounce feeding bottle. This would give dilutions of anywhere from 1:32 to 1:64 if a teaspoonful of condensed milk were one fluidrachm; but this is far from the case, the viscosity of the milk causing it to adhere to the entire surface of the spoon bowl in large mass. It is, therefore, very difficult to estimate the amount used when so measured, since the quantity taken up depends upon the individual and upon how quickly the teaspoon is transferred from the can to the water before part of the viscid mass slowly drops away. The bowl of the teaspoon plunged into the can and immediately transferred to an ounce graduate will carry with it anywhere from 4 to 6 fluidrachms. If after taking up the milk the spoon is held over the can from two to three minutes, until the milk practically ceases to drip, the amount so transferred will measure f5ij. This last method should be insisted upon when accurate proportions are desired.

One such teaspoonful to each 4	oz. of water (3ij	to 3xxxij)	gives proportions of 1 to 16
" " " " 3 1/4	" " (3ij	" 3xxvii)	" " 1 " 14
" " " " 3	" " (3ij	" 3xxiv)	" " 1 " 12
" " " " 2 1/4	" " (3ij	" 3xx)	" " 1 " 10
" " " " 2	" " (3ij	" 3xvj)	" " 1 " 8

Boiled water should always be used in preparation.

Not only are the fat and proteids lower in the usual dilutions employed, but the fat, even in the best brands, is too low for continued use in feeding healthy infants; while the proteids also cannot be raised by less dilution to the point required for nutrition after the third or fourth month, without making the sugar excessive. In a word, then, condensed milk and water cannot be made to furnish suitable proportions of the elements for proper development. If its use be long continued, although the infant may appear fat from the assimilation of the abundant sugar, it will be constipated, flabby, with little muscle or resistance to acute diseases, and will invariably show more or less evidence of rickets. It owes its popularity among the poor to its cheapness and ease of preparation. Condensed milk, well diluted, often serves fairly well for a short time with very young infants or some of those with disturbed digestions who require low fats as well as low proteids, while easily digesting sugar. Being sterile, it is safer than other milk which is obtainable on long journeys, or in summer in the poorest quarters of some large cities; but a change should always be made as soon as possible to good, fresh, cows' milk, remembering that such milk should at first be much diluted. (See p. 148.) When the use of condensed milk is imperative, the deficiency in fat, which is its worst feature, may be made up by adding fresh cream or giving cod-liver oil. For newborn infants, and those of difficult digestion, one may begin with a dilution of 1:16 and increase the strength by less dilution to 1:10 or 1:8.

#### ARTIFICIAL FOODS.

As a fundamental axiom it may be stated at the outset that no artificial food or patented food can take the place of breast milk or properly



proportioned cows' milk for any considerable length of time without injury to the infant's nutrition. I do not go as far as some and say that they should never be used. Many of them have a place in emergencies, to meet certain definite conditions, and at times in cases of difficult feeding. But there should always be in the mind of the physician a clear understanding of the purpose which they are to serve, and a knowledge of what they contain. Nothing is more fraught with disaster to the infant than a trial of first one and then another "infant food" in rapid succession in the blind hope that some one will succeed. Commercialism leads many a manufacturer to claim for his particular food the credit which is really due to the cows' milk with which it is diluted when prepared. Roughly speaking, "infant foods" are (1) preparations of starchy cereals to be added to milk; (2) preparations of soluble carbohydrates (malt and other sugars) to be added to milk; (3) cereal starches with malt and other sugars mixed with pulverized condensed milk. Without the addition of milk neither of the first two classes furnish the materials for full nutritional development. The latter or third class cannot be made to contain a proper amount of fat, and has many of the disadvantages common to condensed milk. Those containing malt-sugar largely are laxatives, and while we may often avail ourselves of this property by adding them to the food of constipated infants, the same property explains the loose stools occurring at times in infants who are given this type of food, the cause of which is often unrecognized. While these sugars are at times better assimilated than milk or cane-sugar, and cause increase of weight by the production of fat, unless the infant foods are to be combined with a suitable amount of fresh milk they cannot be long continued without danger of scurvy and rachitis, and, still more insidious because perhaps concealed by the fat, a poverty of muscular tissue, due to lack of sufficient proteid material, which renders the child exceedingly vulnerable to any intercurrent disease.

Some of those most used in this country are as follows:

1. *Chiefly unchanged starch.* Robinson's Patent Barley, Hubbell's Prepared Wheat, Ridge's Food, Imperial Granum.

2. *Containing no unchanged starch, but large amounts of soluble carbohydrates.* (a) Largely maltose—Mellin's Food; (b) Maltose and other soluble carbohydrates plus evaporated milk—Malted Milk, Cereal Milk; (c) Chiefly lactose—Lactopreparata.

3. *Containing both unchanged and changed starch.* Carnrick's Food; (a) Plus evaporated milk—Nestlé's Food; (b) Largely dextrose and lactose—Eskay's Food.

#### MILK LABORATORIES.

In many of our large cities there exist to-day milk laboratories which undertake to fill physicians' prescriptions for the feeding of any particular infant and to deliver each day the requisite number of bottles containing the amounts and proportions ordered by the physician, ready



for use during the succeeding twenty-four hours. The milk used is derived from model dairies supervised by bacteriologists and veterinarians, and is produced as nearly as possible under ideal conditions for the purpose for which it is to be employed, every care being exercised to secure a clean, fresh milk from absolutely healthy cows. The laboratory does not prescribe; it simply fills the physicians' prescriptions with the best obtainable material, with the nearest possible approach to exactness of proportions and percentages, and with studious care of each step in its preparation, preservation, and subsequent transportation. The following is the usual form of prescription blanks:

PER CENT.		REMARKS.
B-Fat . . . . .		Number of feedings.....
Milk-sugar . . . . .		Amount at each feeding.....
Whey proteids . . . . .		Infant's age.....
Caseinogen . . . . .		Infant's weight .....
Total proteids . . . . .		Alkalinity. ....%
Total solids . . . . .		Heat at.....° F.
Water . . . . .		
100.00		

Ordered for .....

.....

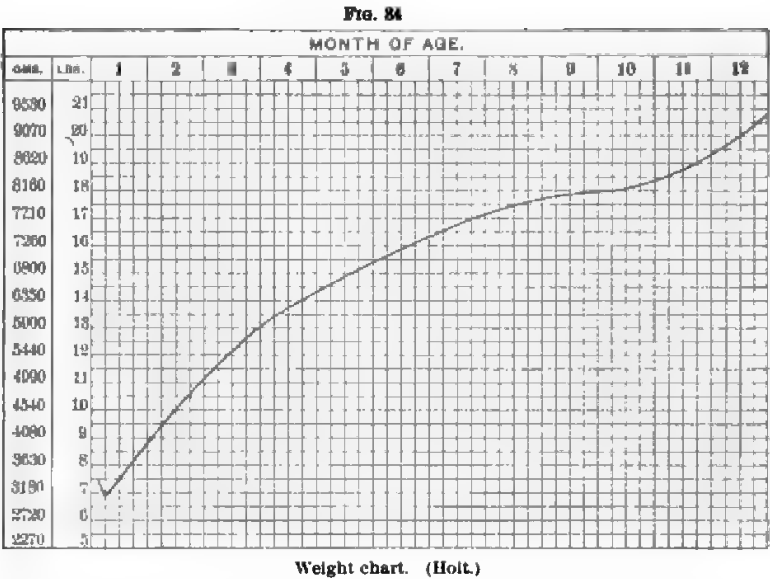
Date.	Signature,
.....190	.....

While at first planned chiefly to secure exactness in the percentages of modified milk and in the quantities furnished, the system has been developed so that the physician may indicate and secure the use of either centrifugal or gravity cream; alkaline, cereal, or whey diluents; milk-sugar or cane-sugar, and may even, if desired, include the addition of proprietary foods. The bottles may be unheated, pasteurized, or sterilized, as preferred. The necessary calculation of the required proportions are made at the laboratory. The prescriptions may be varied within reasonable limits and as often as indicated by the requirements of the infant. It has the great advantage of exactness and ease in varying the amount of any particular constituents of the mixture, some combinations being possible which cannot be secured in the home, and it is consequently adapted especially for the management of cases of difficult digestion. It also relieves the mother and attendants from all responsibility and labor in the preparation of the food, and the physician from giving minute directions concerning quantities and the details of preparation. On the other hand, it presupposes on the part of the physician an accurate and complete knowledge of the exact percentages and

constituents of the food which will be best adapted to the particular case in hand. It is, therefore, suited especially to the uses of the men already well trained in the principles of infant feeding, and has been employed successfully in many thousands of cases when intelligently directed by the physician, upon whose wisdom, in the main, the results depend. Such feeding is naturally expensive, costing usually thirty or more cents per day, and is, therefore, available ordinarily only for well-to-do people. The objections and difficulties arising from this method have been largely due to unintelligent use of its facilities. It is not to be expected that every child will do best upon a laboratory product. There are some failures which thrive with a change to home modifications, and this may have been especially true, as claimed, when formerly only centrifugal cream was used in laboratory modifications; but there are many infants with whom simply a change of method achieves success, and this may be that from careless home modification to careful laboratory feeding. Much of the present success of home modifications is due to the pioneer work of the laboratories in the production of pure milk and in the development of methods of modification.

WEIGHING AND CHOICE OF SCALES.

Much important information concerning the progress of the infant is to be gained from systematic use of properly constructed scales.



Newborn infants and difficult feeding cases should be weighed every second day or twice a week and the weights recorded; for other infants once a week will suffice. While gain in weight does not invariably

indicate well-rounded nutrition, as in infants who receive the high sugar percentages of condensed milk, it is still one of our valuable guides as to the digestion and assimilation of the food. A steady gain in weight even with the simpler forms of "spitting up" the food or moderate disturbance of the stools is reassuring and shows us that with suitable changes these will be readily overcome. Failure to gain in the absence of any disturbing influence and with good stools indicates an increase in the quality or strength of the food. On the other hand, stationary weight or a loss, with disturbed digestion and poor stools, may call for a radical reduction until digestion is re-established.

The chief exception to this latter statement is in children who have been too long upon very low percentages which do not furnish sufficient nourishment, but such cases require careful investigation and good judgment. The usual progress of a normal, healthy infant is best indicated by the accompanying weight chart, devised by Dr. L. E. Holt (Fig. 34). The normal infant should show on the average a weekly gain of not less than four ounces during the first six months; thereafter it may be somewhat less. Infants who are bottle-fed from birth often begin to gain more slowly than the breast-fed, but if good digestion is maintained should later regain the difference. All spring and dial scales are notoriously unreliable and useless to record small variations. To give one any immediate assistance in judging of the effects of the food prescribed, upon children of difficult digestion, the scales should be of the balance variety and register half-ounces. The ordinary grocer's scale with scoop, or scoop and platform, can now be obtained cheaply enough to have a place in every physician's office and to be purchased by parents of even moderate means (Fig. 35).<sup>1</sup>



Scales for weighing infants.

#### FEEDING AFTER THE FIRST YEAR.

Because weaning now becomes necessary at an earlier age than was formerly the case, owing to the failure of the supply of breast milk, especially among the dwellers in large towns and cities, and also generally among the wealthier classes, no ground exists for assuming that the digestive powers of the infant have taken on correspondingly earlier development. It is an unquestioned fact that many children are seriously

<sup>1</sup> A reliable scoop and platform scale of this pattern, weighing from  $\frac{1}{2}$  oz. to 244 lbs., can be obtained from the Metropolitan Hardware Co., cor. Church and Vesey Streets, New York City, at a cost of \$1.00.

handicapped and exposed to great dangers under the mistaken idea that this period is the proper one for the introduction into the child's dietary of general table food. Second only to the revolution in the feeding of the first year by the introduction of modified milk have been the rapid changes of view concerning the necessary elements of the dietary during the second twelve months. With respect to all details pediatricians are by no means fully in accord, but there is a strong tendency on all sides to simplify the diet and to postpone until the latter part of the second year many articles which were formerly given earlier. Much of this diversity of opinion is due to the fact that children at this time, almost as much as during the first year, differ markedly in their digestive abilities, this difference being due to previous methods of feeding, environment, etc. Certain principles have, however, gained general acceptance. The first of these is that milk is not to be abandoned or even allowed to become of secondary importance, but continues the basis of the nourishment until at least the middle of the third year. Where the infant's digestion of proteids allows it is often possible and best to gradually bring it about that at the twelfth month the infant should begin to take plain milk without modification or dilution, save that which results from the addition of cereal jellies. Not all infants can do this, especially those whom digestive disturbances force us to consider and treat as younger than their actual age in months. Some of these must receive modifications of milk the first half or even the whole of the second year. The increased demand for proteids at this period must not be neglected; and if they cannot be taken in the form of milk casein the deficiency should be made up to them in other ways. While the danger of overfeeding the infant at this time more commonly confronts us, we occasionally see infants who, for one reason or another, are continued upon weak formulæ to the detriment of their nutrition, which imperatively demands different and more varied food. The intelligent physician will not attempt to feed all children alike during their second year, any more than he would so feed them during their first year, else he will meet with frequent failure. The age in months does not necessarily furnish any exact criterion of their digestive capabilities. Children reach their second year differing widely in their weight, robustness, nutrition, and their powers of digestion. Some have been nursed and are having their first experience with other food. Others have learned to digest cows' milk months earlier or even from their birth. Some have been brought to this period with the utmost difficulty, and the exercise of the greatest intelligence and ingenuity in the adaptation of the proportions of their food. Certain children of a year old must be treated as though they were many months younger. The tissues of some are starved for the proteids which they have been unable to assimilate sufficiently from such modifications of milk as they could take without disturbance. Still others are prone to starchy indigestion if very moderate amounts be exceeded, or that which is given be not most carefully chosen and prepared, or even subjected to the action of ferments which alter its character. It is with this knowledge in mind



that we should approach the question of feeding the individual child during the second year, and for this reason it is difficult to lay down hard-and-fast rules which shall not be subject to many exceptions. This explains to some extent the diversity among the diet lists of our best authors for this period. Coincident with the demand for more proteid, which is satisfied, in part at least, by the giving of as nearly as possible plain milk, occurs a decreased need of sugar as such, which from now on the body is prepared to make for itself by the action of the salivary and pancreatic secretions upon starch.

**Cereals.**—Whether we allow or not that cereal decoctions, used by many during the first year of life as diluents for cows' milk, undergo much true digestion of their contained starches there is no question but that by the beginning of the second year the amylolytic or starch digesting functions are sufficiently developed to demand this addition to the dietary. Such addition is best in the form of thoroughly cooked and strained oatmeal-, barley-, or wheat-gruel, the thickness of this gruel or jelly varying inversely with the amount to be added to the milk. Their preparation is much simplified by the use of prepared flours. If the grains of barley or oatmeal are used they should be cooked no less than three hours to soften and burst the cellulose envelope which surrounds the starch grains. Those preparations of oatmeal whose grains have already been subjected to rolling, crushing, or steaming give the best results, although requiring much the same amount of cooking, and this necessity for prolonged boiling of all the cereals continues throughout childhood. After cooking they should be strained to remove the coarser particles, and salted. Thorough cooking and straining add much to the digestibility of cereals, and where, despite this, there is still difficulty, it may be often overcome by the addition while hot of a small amount of a glycerinated solution of diastase (cereo), which transforms some or all of the starch into soluble carbohydrates. Barley flour contains less starch, and is more easily converted into sugar by the transforming ferments of the body, and may be chosen when these functions are less developed or weakened. It is to be chosen when there is a tendency to diarrhea. Oatmeal contains more fat, starch, and proteid; so that it is more nutritious than barley, slightly more difficult of digestion, valuable for its laxative properties in constipation, and to be avoided in diarrhea, eczema, and intestinal indigestion. The valuable properties of wheat flour have, to some extent, been lost sight of, except as incorporated in special preparations or used as the basis of a dextrinized gruel. It is more commonly used in the form of stale bread. When cereals are eaten by themselves the most suitable are oatmeal, farina, wheatena, hominy, and cornmeal; always thoroughly cooked and usually strained. Of these, if the child likes it and it agrees, oatmeal is probably the best for the morning meal. Milk or equal parts of milk and cream may be served with the cereal, which should be properly salted, but the use of sugar should be prohibited. Rice, boiled or steamed until each grain is well cooked, may be given alone, or served as an addition to soups and broths, and is, perhaps, more suitable for the midday or evening meal.



**Bread.**—Bread should always be stale and may be well dried also in the oven. Broken into crumbs it may be given moistened with broth or beef-juice or mixed with soft-boiled egg. When there are sufficient teeth the child may be allowed a crust to nibble. It thus earns to chew, and the secretion of the salivary glands is stimulated. Among the poor, bread is given early to children and constitutes their introduction to starchy food, taking the place of cereals combined with milk. Given thus in moderation it fulfils a definite role and is beneficial, but in too large amounts may not be properly digested. "Bread and milk" has a time-honored place as the supper of somewhat older children, but will not always agree as well as when taken separately. Zwieback, unsweetened, is one of the best forms for early use, and is often retained and digested when other foods fail. Italian bread-sticks are also valuable. Various types of plain unsweetened crackers (biscuit) may be allowed later for variety; and gluten, bran, and graham crackers when there is constipation. These latter, however, are frequently oversweetened and cause fermentation. Sweet crackers and lady fingers often cause disturbance and should not be allowed even occasionally.

**Meat and its Derivatives.**—The earliest available of these is beef-juice, the red juice squeezed from a bit of round steak lightly broiled on both sides. This has its strong partisans and opponents, and, as in most such matters, there is a rational middle ground. It is useful even toward the end of the first year, especially for bottle-fed children whose proteid percentages have necessarily been low from any cause or who show any tendency to rachitic or scorbutic changes. It is a blood builder of value for anemic children, and is a tonic stimulant. It should not, however, be given indiscriminately to all children. It is often better withheld from the children of nervous, rheumatic, or gouty parents, who themselves have a tendency to a nervous temperament and to strongly acid urine of high specific gravity or to eczema of the skin. These are often better without it, and often without meat or soups. Some deem it wise to give finely scraped (not minced) pulp of rare beef, one to three teaspoonfuls, where others would use beef-juice, and with good reason, since it contains relatively more proteid and less extractives. On the whole, however, there is a tendency to reserve meat until the latter part or end of the second year, when the presence of the molar teeth indicate greater readiness for food which requires mastication. Mutton, lamb, beef, and white meat of chicken may then be given finely minced, and the child taught to chew them well before swallowing them. This they may not do without watching and instruction. Well-made clear soups and broths are often the first additions after the cereal jellies. Mutton and chicken are preferable to beef for broth, although an occasional use of the latter will give variety. They should be thoroughly freed from fat by cooling, which enables the fat which arises to the surface to be more readily removed. Broths may be introduced into the dietary between the twelfth and fifteenth months, usually at the midday meal, and should be clear, except possibly for the addition of stale bread-crumbs in the earlier months and thoroughly cooked rice in the later

months of the second year. When the family history or symptomatic peculiarities of the individual child contraindicate meat products, milk, soups—*i. e.*, thin purées of peas, beans, cauliflower, asparagus, or celery—may replace those made of meat stock which contain extractives in considerable amount.

**Eggs.** The white of the raw egg furnishes a readily assimilable form of proteid, which is often useful to supplement the proteids of milk, and even for short periods to take their place in emergencies. With meiotic infants the white of one egg may be added to one of the daily feedings. Beaten into 10 to 16 ounces of water and strained with the addition of salt to assist osmosis it is often retained where other food is vomited, and furnishes a bland food in acute disturbances. Although inferior to farinaceous gruels in cases of summer diarrhea, it may be given where the former are refused by the child. Soft-boiled eggs (two minutes) are usually begun at some time during the second year, giving one-half at first. They should be rarely given oftener than on alternate days, as children tire of them easily. The form may be varied by mixing them with stale bread-crumbs, and salt may be added, but no pepper or butter. The yolk of the egg contains tissue-building material of value, including 10 per cent. of lecithin, which enters largely into the formation of the nervous system, and is at times used to supply the deficiency of lecithin in cows' milk as compared with breast milk. For older infants poached or dropped eggs may be allowed for variety, but other forms of cooking render them less digestible and should not be employed.

**Fruits and their Juices.**—Of these, strained orange-juice stands pre-eminent, and is almost a necessary part of the diet of the artificially fed child even during the latter part of the first year, and certainly during the second year. It has valuable antiscorbutic properties, being a specific for infantile scurvy, and since most bottle-fed children have something of this tendency from the nature of their food, better results are obtained by its routine use once a day. It is also a valuable laxative where the tendency is to constipation. In the third year the pulp may be removed and given with a spoon, but the minute sacs which contain the juice will appear in the stools unless the envelope be broken by chewing. The coarse, white fibre should not be allowed. When oranges are not obtainable, the juice of fresh peaches or of ripe fresh berries, strained, may be cautiously tried as a substitute. The fruit element may also be supplied, especially where there is constipation, by the pulp of two or three prunes cooked without sugar and passed through a sieve. Thoroughly cooked apple-sauce or, even better, the pulp only of a well-baked apple is useful toward the end of the second year. Bananas, berries with their seeds, and raw apples are not suitable for the earlier years of childhood.

#### DIET FROM TWELFTH TO FIFTEENTH MONTH.

During this period the child should receive five feedings at approximately the following hours, according to the convenience of the house-

hold: First feeding, 6 to 7.30 A.M.; second feeding, 10.30 to 11 A.M.; third feeding, 1.30 to 2 P.M.; fourth feeding, 5.30 to 6 P.M.; fifth feeding, 9 to 10 P.M.

From 10 to 12 ounces may be given in the bottle at each feeding, and this may consist of from 1 to 3 ounces of cereal jelly or gruel with the remainder plain milk. It is generally accepted that an infant will drink more milk from the bottle than from a glass or cup, and with less effort, often taking from 10 to 14 ounces when it would be difficult to give more than 8 ounces from a glass; therefore, it is well to be in no haste to give up the bottle, which may be continued until the middle of the year, after which some of its contents taken with other food may be poured into a cup in order that the child may learn to drink in that way. The late evening (9 to 10 P.M.) bottle may be continued the longest or until the fifth meal is abandoned.

If the child can take plain milk without dilution, this may, for the sake of variety, replace the milk and cereal at not more than two of the meals, especially when other food is given at the same time. When the time for such additions arrives, either the first or the second meal may constitute the breakfast, according to convenience in its preparation, the third the dinner, and the fourth the supper. The remaining morning feeding and that in the late evening should then consist only of the milk and cereal contents of the bottle. The juice of half an orange should be given each morning at least one hour before a feeding, or, if not obtainable, the juice of a ripe peach may be substituted. Many healthy children thrive on this diet of plain milk, cereal, and fruit-juice and require no other addition. Children of poorer nutrition who cannot digest plain milk, or those long fed on the bottle and showing even slight rachitic symptoms, require other forms of proteid which they may be able to assimilate more readily than those of milk. These may receive once daily the white of one egg mixed with the contents of the bottle and freed from stringy masses by straining, or one to four tablespoonfuls of beef-juice given at the midday meal, or two to four ounces of chicken-broth or mutton-broth. Dry bread-crusts or zwieback may suit the requirements of some children while not necessary for others. The above range of articles may only be exceeded upon definite indications.

#### DIET FROM FIFTEENTH TO EIGHTEENTH MONTH.

The same number of feedings should usually be given during these months. Children who have required up to the fifteenth month only milk and cereals may now receive the additions to their diet provided for in the previous section. For the others a soft-boiled egg may be given twice or three times a week at a morning or midday meal, and the dry bread and zwieback may be increased if already included in the dietary. Thoroughly cooked rice may be allowed in the broth. A further range of fruit juices may be obtained by using those from ripe berries.



**DIET FROM EIGHTEENTH MONTH TO TWO YEARS.**

The child should now be able to eat from a spoon and have been taught to drink some of its milk from a cup. Plain milk may replace that previously given with cereal jelly, and a moderate amount of strained cereal over which milk or equal parts of milk and cream have been poured given at breakfast and supper, farina alternating with bread and milk at the latter meal. Scraped-meat pulp may be replaced by finely minced meat when dentition is sufficiently advanced. Apple-sauce or the pulp of baked apple or stewed prunes once a day. Toward the end of the year spinach, stewed celery, green peas, and string beans, each of which have been run through a colander, may be tried one at a time, as well as a part of a mealy baked potato seasoned with salt or butter or moistened with broth or meat-juices. In my opinion the late evening bottle (9 to 10 P.M.) may well be retained throughout and possibly beyond this period, as the additional nourishment makes it possible to give simpler meals during the day, since the child will often take no more than six to eight ounces of milk at meals where it receives other food, but when the child has attained to a fair range of diet this may be discontinued and the number of meals reduced to four. The season of the year, the child's digestion, and the amount of out-door exercise should influence us in increasing or decreasing the dietary. In the hot summer months, especially, care is necessary, and the less the digestion is taxed the less the liability to serious disturbance.

During the eruption of teeth, if the nervous system is perturbed, the diet should be simplified, since more time and ground may be lost by a digestive upset than would be gained by pushing the food. Foul-smelling stools or those containing undigested food give us warning, and call for a return to a simplified diet or even to milk alone for a time without waiting for more serious symptoms to develop.

Desserts had better be withheld until the third year. If given at all they should consist of junket, unsweetened custard, cornstarch, and plain rice-pudding without raisins. The diet already outlined gives ample variety for a child's needs. Children whose digestions are kept in good condition do not need constant changes to stimulate appetite and are satisfied with a simple régime. Children should not be allowed to know the taste of candy and sweets. They will elaborate from their starches all the sugar they require, and it is the beginning of endless trouble when they realize the existence of articles which taste better than their every-day food. Mothers are constantly inquiring whether a little of this or that would hurt the baby. The difference between the plain fare which can be taken safely and those things which might not cause disturbance is a wide one—between lies debatable ground upon which it is folly to tread. The giving of tastes of this or that food has its true basis quite as much in the selfish gratification of the giver as in the pleasure of the child. For this reason young children should, if possible, be given their meals by themselves and not at table with

adults or older children, for in the latter case they will inevitably secure unsuitable articles either by stealth or through importunity. Children may often be induced to eat more of certain plain but nourishing dishes by giving these first while they are hungry and reserving those for which they show the greatest fondness to be placed before them at the end of the meal.

#### **DIET DURING THIRD AND FOURTH YEARS.**

The articles already mentioned give a sufficient range until a child is two and one-half years of age, and in practice it is often wisdom to wait until the third year before giving meat, many of the above vegetables, and preferably any form of dessert. By the thirtieth month the range of well-cooked cereals may be increased. Absolutely fresh fish of firm white meat, mashed cauliflower, squash, and strained tomatoes may now be tried. Among the fruits the pulp of fresh, ripe pears and peaches may be allowed in small amounts. Raw apples and bananas and the small seed berries should be barred as sources of danger, but the juice of the latter may be allowed as previously. Plain vanilla ice-cream is permissible, but not exceeding twice a week. Definite hours for meals should be established, and the pernicious habit of eating between meals discountenanced.

#### **DIET FROM FIFTH TO EIGHTH YEAR.**

This is still an important period over which a sufficient supervision is rarely exercised. During this time the child lays an almost equally important foundation for future years, although the immediate danger from digestive disturbances is vastly decreased. The habit of eating simple, nutritious food in good variety should now be formed. The natural preference for sweet articles to the exclusion of plain food should be combated by withholding the former. A child should never be coaxed to eat by the introduction of jams, jellies, preserves, syrup, or candy. If the child fed on plain food shows a continued lack of appetite it requires medical attention. It is a common experience to find thin, poorly developed, and often anemic children with coated tongues and without appetite, in whom both appetite and nutrition may be restored by absolutely forbidding all candy, sugar, and sweet foods, and giving some simple tonic, such as the bitter wine of iron, after meals. No food should be given except at meal-time. Dry, canned, smoked, salted, and preserved meats and foods should be avoided. Tea, coffee, beer, and even cocoa are unnecessary and have no place in the dietary of children, as also highly seasoned and made dishes, cake and sweet desserts. It is a good rule that they should have only such plain food as would be allowed a convalescent adult. They will thus escape with fewer acute illnesses, and approach puberty with sound digestions, better physiques, and normal appetites.



## SECTION IV.

### DISEASES OF THE ALIMENTARY TRACT.

By DAVID BOVAIRD, JR., M.D.

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#### CHAPTER IX.

##### DISEASES OF THE MOUTH AND PHARYNX.

##### DISEASES OF THE MOUTH.

##### CATARRHAL STOMATITIS.

THIS form of inflammation of the buccal mucous membrane and tongue is most common to the first year of life, but is not uncommon in the later periods. Often it is an independent affection, produced by uncleanness or lack of care, or by some form of irritation, chemical, mechanical, or thermal. It regularly occurs with any febrile affection that is prolonged for many days, and especially with such as are due to inflammatory disturbances in any part of the alimentary tract, particularly the diarrheal diseases. It is observed in children who are fed improper food. It may occur in the eruptive fevers, but it is certainly stretching the conception of a catarrhal affection to make it include all the varied changes in the buccal mucous membrane that occur in measles, scarlet fever, and like affections. Local lesions, especially decayed teeth, may produce a catarrhal stomatitis, and I believe that I have seen it produced by the continued use of the popular pacifier.

In some cases dentition may excite this process. Infection may possibly be concerned in the etiology, but the mouth is such a storehouse of bacteria that it is difficult to identify any as the excitants of disease. More or less general catarrhal stomatitis is regularly associated with the presence of the severer types of inflammation of the mouth, ulcerative, aphthous, etc.

**Lesions.**—The mucous membrane of the mouth is congested, the finer capillaries are dilated, and in the severer cases there may be minute hemorrhages into the tissues, or bleeding may be produced by slight traumatism. The mucous membrane may be swollen and the tongue may appear correspondingly large and thick. The dorsal surface of

the tongue is regularly coated with a white, yellowish, or brown fur; the edges are red.

The normal secretion of the mucous membrane is arrested in the earlier and increased in the later stages of the affection. The duration of the disease is determined mainly by the nature of the exciting cause. As an independent affection catarrhal stomatitis will run its course within a week. When associated with the fevers it may persist until the underlying condition mends.

**Symptomatology.**—The symptoms comprise those of any catarrhal inflammation of a mucous membrane—redness, swelling, pain—and either a diminished or increased secretion of mucus. There is usually an increased local temperature. The soreness of the mouth may be slight or may be severe enough to seriously interfere with the feeding of the child. The child may be very fretful and peevish and refuse nourishment because of the pain. In protracted fevers the condition of the mouth may be for this reason an important factor in determining the outcome of the disease. Constitutional symptoms are not commonly seen, but a slight rise of temperature may be noted, and vomiting may occur. On inspection we can see the reddened, swollen mucous membrane, possibly showing minute hemorrhages, either dry and glazed or bathed in an increased secretion of mucus. In the later stages the membrane, particularly that of the tongue, may be cracked or deeply fissured, and covered with a deposit of thick mucus and exfoliated epithelium. There is not any involvement of the lymph nodes, as a rule, but in the severer cases they may become slightly swollen and tender.

**Diagnosis.**—The local appearances are characteristic. The most important point in the examination of the mouth is to determine the presence or absence of any causative factor, such as decaying teeth, or of other and more serious lesions, such as aphthæ, ulcerations, etc. Thorough examination is very important, and to secure this one must know how to handle children. For a satisfactory examination of the mouth in an infant or young child the nurse or mother should stand with the child and turn it to face the light and the physician. The mother should with one arm clasp the legs and with the other the hands, so that the child's head falls on her right shoulder. The physician can then control the head with his left hand, while with the right he uses a spatula or spoon to open the mouth, depress the tongue, retract the cheeks, etc. For the purpose of a spatula the little flat sticks adopted by the Department of Health of New York are most satisfactory. They will not slip as the polished spatula does, and are so inexpensive that they may be thrown away or burned after using, as the nature of the case requires.

**Treatment.**—For the primary cases the removal of the cause, if discoverable, may be all that is required. Usually some local treatment is necessary. In the febrile affections, and particularly the eruptive affections and diarrheal diseases, the care of the mouth is of very great importance, because of the interference with feeding which results from

the stomatitis, and the possibility that a complicating pneumonia may be caused by neglect in this particular. Thorough cleanliness and the use of some mild antiseptic wash to prevent decomposition of the normal secretions are essential, both in prophylaxis and in treatment of catarrhal stomatitis. After each feeding the mouth should be cleansed with a 2 per cent. solution of boric acid or some equivalent. There are a number of proprietary mixtures which are very serviceable in this regard, because they meet the indications and are at the same time agreeable to the patient. Among these may be mentioned glycothymoline and borolyptol, each used in a dilution of 1 part to 4 of water. The nurse should swab the mouth with one of these solutions, using for the purpose absorbent cotton wound upon the finger or a small stick. Gentleness should be required, as injury is readily done by rough usage. In the severe or protracted cases it may be necessary to use some astringent application, such as a weak (2 per cent.) solution of nitrate of silver, which may be painted over the mucous membrane with a camel's-hair brush once a day for several days in succession. Alum in a 2 per cent. solution may be used in the same way, or powdered alum may be mixed with an equal quantity of bismuth and dusted over the surface, where the process is protracted or superficial ulcerations have formed.

The feeding of the patient is of importance. The food will produce less pain if given cold. If the child altogether refuses to feed in the normal manner it may be fed by gavage as often as may be deemed necessary. It is not usually advisable to attempt to keep up a program of feeding every two or three hours in this manner. Three or four feedings in a day may, however, be given with the tube.

#### APHTHOUS STOMATITIS.

Aphthous Stomatitis is also called Aphthæ, Herpetic Stomatitis, Herpes, but the first designation is most descriptive.

**Etiology.**—On this point there is little known. The affection is relatively rare among infants in their first year, but is common during the second and later years of childhood. Many authors regard it as of nervous origin. French writers especially consider aphthous stomatitis as an infectious and contagious disease identical with the foot-and-mouth disease of cattle and transmitted from cattle to children by means of the milk, but the evidence of this relationship is not satisfactory. The French also describe herpes of the mouth as a distinct and independent affection. Aphthous stomatitis, as we see it, is regularly associated with digestive disorders, but whether as cause or effect we are not prepared to say. There is no evidence of its communication from one child to another.

Aphthous stomatitis is often associated with dentition. Baginsky thinks that it is especially frequent in children living in damp, newly built houses or in badly ventilated dwellings. He also says that it may

occur in several members of a family, but that evidence of its transmission from one to another is lacking. Forchheimer and others consider the affection as analogous to herpes. The variety of views regarding its nature is sufficient evidence of the incompleteness of our knowledge.

**Lesion.**—In its earliest stages this is a vesicle, formed by an exudation between the superficial epithelium and the underlying mucosa. The epithelium is quickly destroyed and there is left a superficial ulceration, small, round, not indurated, ringed about with a narrow zone of bright-red congestion. The floor of the ulcer is at first yellowish, its diameter commonly 3 to 4 millimetres; in its later stages the color may become a dirty gray. The ulcers are usually scattered, are most numerous upon the tongue and inner surface of the cheeks, but may occur upon any part of the mucous membrane of the mouth or pharynx, even upon the tonsils. Occasionally they become so numerous as to fuse into one another and form ulcerations of considerable extent, but still superficial. In the cases commonly seen there are not more than six or eight ulcers in the mouth. Various bacteria have been found in the lesions, but no specific relation has been demonstrated between them and the lesions of the disease.

**Symptomatology.**—With or without fever the characteristic lesions appear in the mouth. Usually for several days there is an eruption of new spots. The gums are swollen and the whole mucous membrane is deeply injected. The tongue is heavily coated white. There is a profuse salivation. The ulcers are extremely painful and the children are consequently restless, fretful, and refuse to eat. The affection tends naturally to recovery in from one to two weeks.

**Diagnosis.**—This must rest upon the characters of the local lesion. The round form, discrete distribution, superficial character of the ulcers, together with the bright-red ring of congestion about them, are distinctive. The fetid odor of ulcerative stomatitis is never present, although the breath may be heavy.

**Treatment.**—This is essentially the same as that of the catarrhal stomatitis, except that the pain can be considerably relieved and progress hastened by touching the ulcers with a lump of alum or by dusting powdered burnt alum upon them. In protracted cases nitrate of silver may be used. Peroxide of hydrogen diluted with 3 or 4 parts of water forms an excellent mouth wash or application for cases of this kind. Instead of the alum, potassium permanganate 1:15 may be applied with a brush to the lesions daily for several successive days. If these applications do not sufficiently relieve the pain, cocaine in 2 per cent. solution may be similarly applied to the lesions, but this is usually unnecessary.

The internal administration of potassium chlorate or other remedies may be dispensed with. As already noted, the disease is usually self-limited. The object of treatment is mainly to relieve the discomfort.

At the onset a dose of castor oil or calomel is advisable to move the bowels freely. Milk of magnesia is a useful laxative. The diet should be milk modified to suit the digestive powers of the child.



**ULCERATIVE STOMATITIS.**

This affection is never seen in infancy before the eruption of the teeth. It is common after the second year of childhood, and it may occur at any age. In the great majority of cases it is to be attributed to bad hygienic conditions. It is rarely seen in private practice, but is extremely common in children confined in hospitals and asylums. It is a frequent sequel of infectious diseases, especially measles, typhoid, pneumonia, etc. It is this form of stomatitis which is a regular accompaniment of scurvy, and doubtless defects in diet are responsible for the frequency of its occurrence in hospital and asylum children. Ulcerative stomatitis may be produced by the excessive use of mercury, iodine, lead, or phosphorus, but except from mercury we rarely see it caused in this way. It is held by some to be a contagious affection. Forchheimer says that under proper conditions it can be transmitted, but, as a matter of practical experience, contagion appears to play no part in its distribution. Lack of care of the mouth and teeth, and especially neglect of decaying teeth, seem to be important factors in the production of this affection. No specific organism has yet been found.

**Pathology.**—There is an intense general catarrhal stomatitis, the gums especially being swollen, so that they may almost cover the teeth. They are deep purple in color and bleed very easily. The characteristic lesion is that of necrosis and death of that portion of the gums which extends upward on the teeth. This part of the gums at first shows a yellowish line of necrotic tissue; later, the dead tissue becomes dark gray or black and sloughs off, leaving a raw, bleeding surface. There is a mucopurulent exudation between the gums and the teeth, which are loosened in their sockets and may even be entirely detached. Similar death of tissue and ulceration may appear on other parts of the mucous membrane of the mouth, especially on the inner surface of the lips and cheeks opposite the teeth, but the ulceration never extends beyond the limits of the mouth. The submaxillary and anterior cervical lymph nodes are regularly swollen and tender.

Cornil and Ranvier describe the pathological process as a diffuse infiltration of the lymph spaces of the tissue with pus and fibrin, by which the capillaries of the part are compressed, and the circulation checked, so that the death of the part follows exactly as in a phlegmon of the subcutaneous tissues.

The ulcerations produced on the mucous membrane are irregularly round in outline, moderately deep, the edges ragged, the base a dull gray or grayish black, covered with thick mucus of an extremely fetid, offensive odor. The ulcers are of any size, often becoming extensive.

In severe cases the periosteum of the jaw may be involved and there may be extensive necrosis of the bone, sequestra of considerable size being removed.

**Symptomatology.**—Attention is usually attracted to the condition of the mouth either by complaint of pain in eating or by the profuse



salivation and the very fetid odor of the breath. On inspection of the mouth we usually find the mucous membrane everywhere, but especially the gums, swollen, deep purple, spongy-feeling, and bleeding at the slightest touch, while about the roots of some of the teeth, most often of the lower incisors, but sometimes the molars, there is the characteristic yellowish or gray line of necrotic tissue. Ulceration of the mucous membrane may be found, particularly in the fold passing from the jaw to the inner surface of the lips, or opposite the molar teeth. These ulcers have the characteristics already described. Profuse salivation is noted and from this there may be extensive excoriation and eczema of the lips or skin wet by the saliva. The swollen submaxillary and cervical lymph nodes can be felt.

The affection is extremely painful, the children are restless and peevish and greatly depressed, as a rule, because of their inability to take nourishment. The temperature may be slightly elevated in the beginning, but is usually normal. The offensive fetid odor exhaled by the patient's breath and the saliva is perceptible even at a distance. The patients will hardly touch food or drink, because of the severe pain excited.

In favorable cases the necrotic tissue soon sloughs off, leaving raw, bleeding surfaces, the swelling of the mucous membrane subsides, the ulcers gradually heal, the salivation subsides, and the mouth returns to normal. This is the ordinary course, but under unfavorable conditions the ulceration of gums and mucous membrane progresses, the teeth are loosened and fall out, and the alveolar process of the inferior maxilla may be totally destroyed. The upper jaw, if affected, is much less seriously involved. These graver ravages are not common. The affection usually runs a favorable course, but at times will persist for weeks or months and may seriously drain the child's vitality and impair nutrition. More or less loss in these respects is regularly seen.

**Diagnosis.**—Simple inspection suffices for this purpose. The swollen, deep-purple gums; the necrotic line at the roots of the teeth; the distribution of the ulcers and their character; the profuse salivation, and the fetid odor of the breath are characteristic. The ulcers may suggest diphtheritic lesions, but the absence of a diphtheritic process in the throat and the results of cultures will promptly settle the question if the local appearances are confusing.

At times the question may arise whether we have to deal with ulcerative stomatitis or noma. In noma the tissues have the deep blue-black look characteristic of gangrene, the odor is characteristic, and the constitutional symptoms of high fever, marked toxemia, and prostration are enough to settle the question.

**Treatment.**—In most cases impaired nutrition from improper or inadequate food or bad hygienic surroundings is an important factor and requires attention. An antiscorbutic diet, with fresh milk, fresh fruits and vegetables, will be of help. In the early stages only fluids or very soft foods can be taken.

Locally, we must remove decayed or broken teeth or any other cause of irritation. Teeth that are merely loose may be spared in the hope

that they may be retained, but where necrosis of the alveolar process occurs it will be necessary to remove the affected teeth and scrape away the softened bone or wait for the separation of a sequestrum.

The use of a detergent and antiseptic mouth wash, as in the catarrhal stomatitis, is helpful. If the child is not old enough to use a mouth wash itself, the nurse or mother may be directed to cleanse the mouth after each feeding in the manner described under catarrhal stomatitis. Great gentleness will be required in any such manipulations. Potassium chlorate is regarded as a specific for this affection. It is regularly given internally and may be employed as a mouth wash. It may be given simply dissolved in water in the proportion of 0.130–0.195 gm. to 4 c.c. (2 or 3 grains to the drachm), or in such a prescription as the following:

R <sup>x</sup> —Potassii chloratis.	. . . . .	60 gms.	(3iss).
Acidii muriatici dil.	. . . . .	40 c.c.	(3j).
Syrupi . . . . .	. . . . .	15.0 "	(3ss).
Aquæ . . . . .	q. s. ad	120.0 "	(3iv).—M.

Sig.—2.0–4.0 c.c. (3ss–3j) every two or three hours.

1.30–1.95 gm. (20 to 30 grains) may be given during a day to a child of three years, but such doses should not be continued for more than a few days. The potash has hemolytic powers and is capable of producing an acute nephritis, but such results are very rarely seen. After two or three days the mouth will begin to improve and the doses should be diminished.

As a mouth wash potassium chlorate may be used in the strength of 0.2–0.25 gm. to 30 c.c. (3 or 4 grains to the ounce), but it is rather painful for such purpose. As the mouth improves the diet should be increased and nutrition favored in every possible way. Iron tonics may be employed, but the best of tonics will be fresh air and good food. In the protracted cases caustics, such as alum or nitrate of silver or the potassium permanganate, may be employed to bring about healthy reaction. Necrosis of bone may require removal of teeth and scraping of the alveolar process, or the removal of a sequestrum after separation.

It is to be remembered that much destruction of bone may impair or even destroy the permanent teeth and also lead to considerable deformity from falling in of the soft parts.

#### MYCOTIC STOMATITIS.

This is a specific stomatitis produced by the growth and development of a cryptogam, and it is commonly named thrush. The affection is regularly seen in infants in the first few months of life, although it does occur in rare instances later. It is of frequent occurrence in hospitals and asylums unless great care is taken of the infants' mouths. It is not uncommon in dispensary practice, among the children of the poor and ignorant, but is practically unknown where proper care of the newborn is taken. Artificially fed infants suffering from intestinal disorders or any wasting disease seem to be much more prone to suffer from this affection than breast-fed and the healthy.

The fungus which produces the disease is frequently found in the air, and infection may occur from this source; but the infection may also be carried by unclean nipples, bottles, cloths, or instruments that are used in the mouth of the infant.

The fungus presents itself in filaments 3 to 4 mm. wide and 50 to 60 mm. in length, joined in long threads. At the junctions of the filaments rounded cells branch off and in these spores are found. The spores reproduce the filaments or mycelia. If one of the small whitish patches characteristic of the disease be scraped off and put under the microscope, either unstained or colored by a methylene-blue solution, it will be found to consist of epithelial cells from the tongue, various yeast cells, and the mycelia and spores of the specific fungus. The exact classification of the fungus has given rise to much discussion and is still unsettled. By some it has been considered as identical with the mould fungus, the *oidium lactis*, found in sour milk; by others it is classed with the yeast fungus or wine ferment, *saccharomyces mycoderma*; still others class it as a yeast fungus not identical with the last named and call it *saccharomyces albicans*. From the practical standpoint the question is not of vital importance.

**Lesions.**—The characteristic lesion is a minute white spot, the size of a pin's head, slightly raised, occurring upon the tongue or any part of the mucous membrane of the mouth. The lesions may occur in the larynx, esophagus, stomach, cecum, and even, it is said, in the lungs. They are usually discrete, but may fuse and form considerable areas, until the whole mouth is lined by the whitish coating. The patches are found to be closely adherent, so that they are not easily removed. If scraped off, they leave a red, glistening surface beneath.

In fatal cases in infants we find the lesions of gastric or intestinal catarrh or marasmus. In older children mycotic stomatitis is usually associated with tuberculosis, typhoid, or persistent pneumonia.

The severer types of the disease are evidently, from the literature, much more common in Europe than in this country. In several thousand autopsies at the New York Foundling Hospital but one instance of the finding of the fungus beyond the mouth has been recorded. In that case the fungus was found in the stomach (Northrup).

**Symptomatology.**—Preceding the development of the characteristic lesions of thrush the mucous membrane of the mouth is somewhat swollen, reddened, and dry, as in a catarrhal stomatitis the infant will show the usual distress in feeding and will be restless and fretful. After a day or two the characteristic minute white patches, slightly raised above the surface, dry and adherent, appear, first upon the dorsum of the tongue, later on the mucous membrane of the lips and cheeks, the gums, and the palate. As the patches become more numerous they also increase in size and may fuse into one another until the whole mouth is coated with a whitish pellicle. In the course of a week the patches assume a grayish or yellowish tint, become loosened from the mucous membrane, and are gradually exfoliated, the underlying membrane being left red, dry, and with prominent papillæ. The affection

PLATE IV.



- Fig. I Throat.  
 II Ulcerative Stomatitis.  
 III Acute Stomatitis.  
 IV Acute Stomatitis. Life stage.  
 V Geographical Tongue. Eczema of the Tongue.





then terminates by a gradual return of the membrane to a normal appearance or there is a new evolution of the plaques and a continuation of the disease. During the height of the disease there may be occasional vomiting, loose yellow or greenish stools, and considerable pain, restlessness, and irritability. The temperature may be slightly raised, but is generally normal. As commonly seen the patches are not very numerous, they remain discrete, and the constitutional disturbance is slight. The reaction of the mouth is regularly acid during the course of the affection.

Occurring as a complication of preceding diarrheal disease, or in atrophic children the affection not infrequently assumes a grave type. The mouth becomes coated with the whitish deposit, which may extend into the pharynx, esophagus, and even into the stomach and intestines; vomiting may be frequent, diarrhea severe, with frequent green, acid stools, which excoriate the buttocks and any other parts with which they come in contact; the temperature may be high, the interference with feeding marked; the patients may waste rapidly and the disease terminate fatally.

**Diagnosis.**—The affection can usually be recognized at a glance. The inexperienced may be misled by the presence of little flakes of milk, which may present an appearance not unlike the patches of thrush. The milk flakes are, however, easily brushed off; thrush plaques are, during the early days of the disease, quite firmly adherent. If doubt remains in any case it can at once be settled by scraping off one of the little patches and examining it under the microscope for the specific fungus. The ulcerative affections can be readily distinguished by the destruction of epithelium and the consequent depressions. Thrush is a deposit elevated above the surrounding surface. The lesions of aphthous stomatitis present a superficial ulceration, are yellowish in color and usually ringed with a bright zone of congestion. The salivation seen in these ulcerative diseases does not belong to thrush.

Diphtheritic stomatitis is usually limited to one or more patches, and is accompanied by diphtheritic lesions of the throat.

The microscopic examination is conclusive in any case.

**Prognosis.**—This is usually very favorable. As ordinarily seen the cases are mild and relieved in a few days. It is to be remembered, however, that in children already exhausted by preceding disease thrush may prove a serious and even fatal complication.

**Treatment. Prophylaxis.**—This is of the utmost importance in all cases, and especially in hospitals and asylums. Strict cleanliness of the infant's mouth is the first essential. The mouth should be cleansed after every feeding with a 2 per cent. boric acid solution. The cleansing is best done by the use of absorbent cotton wound on the nurse's little finger or a stick and then wet with the solution. If for any reason this method is undesirable a soft brush may be used as a swab. The next important point is the regular cleansing and sterilization of bottles and nipples used in feeding, and of any instruments or articles that it may be necessary to put into the infant's mouth. After cleansing,

bottles and nipples should be kept in a 2 per cent. boric acid solution until used.

By strict attention to these details the affection has been banished from modern lying-in institutions, but any relaxation of care is quite sure to be followed by an outbreak of the disease.

*Curative.*—The same methods are usually adequate to remove the disease after it has appeared. Instead of the boric acid, 2 per cent. sodium bicarbonate may be employed. The systematic use of these solutions will usually result in a prompt cure. In rebellious or severe cases stronger solutions may be necessary. A saturated solution of boric acid may be applied three or four times a day. Bichloride of mercury 1 : 1000 may be applied once or twice a day.

Where it seems desirable to prolong the effect of the application this may be done by dissolving the antiseptic in glycerin, as in the following:

<b>R</b> —Boracis,									
Sodii bicarbonatis	.	.	.	.	.	āā	4.0 gms.	(3j).	
Glycerini	.	.	.	.	.	.	30.0 c.c.	(3j).	—M.

This solution can be painted on with a brush. For still more powerful effect permanganate of potassium, 1 : 250, or 2 per cent. silver nitrate solution may be employed with a brush. Whatever application is made care should be taken to avoid any further injury to the epithelium. In the severer types of the affection, met with secondarily in the diarrheal diseases, etc., special attention will be required to the feeding, which must be adjusted to the disturbed digestive functions of the infant. Holt says that in certain hospital cases he has found that the disease is sometimes protracted by the irritation produced by the nipple in feeding, and suggests in such cases resort to feeding by gavage for several days.

### PERLECHE.

Under this title an ulcerative affection of the angle of the mouth was originally described by Lemaistre and has since been more thoroughly considered by Comby.

*Etiology.*—The affection is not uncommon among children from two to seven years of age and may be seen in the younger classes of school children. The fissure with which it begins is doubtless produced by traumatism; in the later development of the ulceration the practice of constantly licking the lips and secondary infection by the streptococcus or staphylococcus appear to be the most important factors. The affection may present itself in several members of a family and is thought by some to be contagious, transmission occurring from the use of unclean drinking vessels, etc., or by kissing.

*Lesion.*—This is at first a simple fissure of the angle of the mouth. Later there develops a superficial ulcer with a dirty-gray base, and some thin, purulent discharge. The ulcer is not unlike the mucous patch of hereditary syphilis. Under unfavorable conditions the ulceration may

become extensive and there may be destruction of the surrounding skin. Usually there is no involvement of the lymph nodes, but in the severe cases this may occur. The ulceration is usually quite painful and the lip may be swollen. The affection is not grave, yielding readily to treatment.

**Diagnosis.**—The character and location of the ulceration and the absence of any of the other symptoms of syphilis suffice to render diagnosis easy.

**Treatment.**—This consists in cleansing the ulcer, touching its surface with a caustic, the nitrate of silver stick, tincture of iodine, or burnt alum, and later the application of a protective ointment, such as one of bismuth or zinc oxide.

#### **BEDNAR'S APHTHÆ.**

By this name certain superficial ulcerations, produced by traumatism upon the hard or soft palate, have come to be known. The lesions are undoubtedly produced by rough treatment in swabbing out the mouth and occur just at the points where too great pressure would readily tell. These are well back upon the hard palate, just at the junction with the soft, and over or close to the velum of the palate. They are quite frequently seen in hospital practice, rarely in private work. In some cases similar ulcers are produced by too large or rough nipples. The ulcers are usually round or elliptical, yellowish in color, and very superficial. They are regularly seen during the first weeks after birth. They may be important by reason of resulting difficulty in feeding.

**Treatment** should be that of a catarrhal stomatitis, cleanliness being especially important, and for obvious reasons special care should be taken to avoid further injury to the delicate mucous membrane.

#### **GONORRHEAL STOMATITIS.**

A form of gonorrheal involvement of the mouth, consisting of superficial ulcerations upon the tongue or the palate, has been described by some writers. The infection occurs from the mother. The gonococcus may be demonstrated in the secretions of the ulcers, and this demonstration is essential to the diagnosis. Very little is known of the affection and it appears to be a rare occurrence.

**Treatment** would be that of the catarrhal stomatitis.

#### **SYPHILITIC STOMATITIS.**

Under this heading may be included fissures, papules, mucous patches, ulcers or primary sores. It is quite unusual to find the primary lesion of syphilis in the mouth in children, but it may occur upon the lips, tongue, or tonsil. Its characters and course do not differ from those

observed in later life. Fissures of the mucocutaneous surface of the lip are common and well-known manifestations in congenital syphilis. The fissures are most often seen at the angles of the mouth, but are not limited to that site. They may be deep, they bleed easily, and are painful. There may be some induration about them. They should not be confused with the simple fissures seen in poorly nourished children during or after the exanthemata or other severe illness. The syphilitic fissures are very chronic and difficult to heal. They regularly leave cicatrices which often produce deformities of the lip that are quite characteristic. (See chapter on Congenital Syphilis, p. 562.)

The mucous patches occur upon the lips or any part of the buccal mucous membrane. These are usually round, of a grayish-white color, sharply limited, and slightly raised above the surface. They are not, as a rule, painful. Papules, the condylomata lata, are not frequent, but may occur about the mouth. They are usually broad, their surface irregular, the centres soft and exude a purulent secretion.

**Diagnosis.**—While the appearance of these local lesions is often characteristic, it is certainly unsafe to venture a diagnosis in the absence of other evidences of syphilis, the coryza, adenopathy, laryngitis, or eruptions.

**Treatment.**—The constitutional treatment of syphilis is, of course, essential, and is mentioned in detail in another chapter. Where the local lesion permits, mercurial ointment may be applied to it, or it may be dusted with a powder of equal parts of calomel and bismuth. The mouth should be kept thoroughly clean by the use of one of the detergent mouth washes.

#### DIPHTHERITIC STOMATITIS.

This is one of the possible complications of diphtheria in general. In my experience it is never seen in the absence of diphtheritic affection of the pharynx and tonsils. In the mouth the diphtheritic plaques may occur on any part of the mucous membrane, but especially on the tongue or the inner surface of the lips. In severe cases of diphtheria it is not uncommon to see fissures at the angles of the mouth covered with membrane. The lesions in the mouth have all the varied appearance of diphtheria seen elsewhere. As it regularly accompanies diphtheria of the throat, mistake can hardly be made in the diagnosis. Non-diphtheritic lesions, the ulcerative or aphthous stomatitis covering large areas, are sometimes mistaken for diphtheria. The local appearances ought to decide the matter, but, if necessary, cultures may be made. In association with diphtheria of the mouth the submaxillary and adjacent lymph nodes should be swollen and tender.

The occurrence of lesions in the mouth is of some moment by reason of the resulting pain and greater disinclination to the taking of nourishment, otherwise one should not attach too grave importance to them. More or less catarrhal stomatitis is associated with the diphtheritic lesions.



**Treatment.**—Apart from the use of antitoxin this would call for the local cleansing applied to diphtheria of any part, by the frequent irrigation with normal salt solution or 2 per cent. boric acid. No attempt should be made to remove the membrane mechanically, and more vigorous treatment is usually more harmful than helpful.

### GANGRENOUS STOMATITIS.

This disease is described under the names of Noma, Cancrum Oris, and Wasserkrebs.

**Etiology.**—The affection is not infrequently seen in large hospitals or asylums for children; it is almost unknown in private practice. From time to time isolated cases are, however, reported in children in homes far removed from all the usual influences or sources of infection. Undoubtedly bad hygienic surroundings favor the outbreak of the affection; this is implied in its confinement to hospitals and asylums. Whatever lowers the resisting power of the child favors the disease, but internal conditions are the factors of greatest importance. The disease almost never occurs primarily but is a sequel of some exhausting illness, such as measles, scarlet fever, pneumonia, typhoid, whooping-cough, dysentery, tuberculosis, syphilis, etc. Of all these measles is *par excellence* the precursor of cancrum oris. The active stomatitis which regularly accompanies this affection undoubtedly plays a part in the production of gangrene. The disease is most often seen in children from two to seven years of age, but I have seen it in an infant of six months, and Köster, Hannson, and Ziegler have reported cases ranging in age from fifteen to seventy years. Geographically its special field lies in Denmark, the Baltic coast of Germany, and Holland. The disease is regarded by some as contagious, but the support of this proposition is not strong. Blumer and Macfarlane have, however, reported an epidemic in the Albany Orphan Asylum. In one of the nurseries of the New York Foundling Hospital a few years ago there occurred a series of cases of gangrene of the ear, in which the transmission of the disease was satisfactorily traced to the common use of a syringe for irrigation of the ears. The outbreak stopped upon the introduction of proper methods of asepsis.

**Pathology.**—The bacteriology of gangrenous stomatitis is still in dispute. Schimmelbusch in 1889 described a short bacillus, with rounded ends, occurring sometimes as a diplobacillus, sometimes in long filaments, which he found in the zone of invasion of the gangrene and which he regarded as specific. Rossi in 1892 found streptococci and staphylococci with many leptothrix-like bacilli in the lesions. Babes and Zambiloviei in 1894 described another specific bacillus. Blumer and Macfarlane found an organism of the leptothrix class. Others have found the diphtheria and pseudodiphtheria bacilli, but none of these organisms has satisfactorily been proven to be specific. In the nature of things many organisms will be found in any gangrenous process about the mouth.



**Lesions.**—In a single instance I have seen cancrum oris develop in a child before the eruption of the teeth. In all other cases my experience confirms Monti's view that cancrum oris develops from a previous ulcerative stomatitis. We regularly find the margin of the gums overlying several teeth, usually the molars, dark and necrotic; the teeth are loosened or have already fallen out; if drawn, the roots are found bathed with a thin, greenish-black exudation, which gives the characteristic stench of gangrene. The alveolar process is softened to a greater or less extent and the destruction may extend through the maxilla and, if in the upper jaw, involve the floor of the nasal passages or even of the orbit. In the bone the disease spreads by extension, but never seems to involve the palate to great extent. Some writers state that the process never attacks both sides of the jaw, but in my experience this is not so uncommon. The most striking part of the clinical appearances, the gangrene of the soft parts of the face, is in my judgment a secondary process and is a true gangrene. The margins of the destroyed area are black or grayish black, ragged and sloughing. The discharge is thin, dark greenish black in color, and of characteristic odor. There is no line of demarcation, the color of the gangrenous area fading gradually into that of the normal skin.

Microscopically, in the margins of the affected area sections show a zone of necrosis, in which the general topography of the tissues can still be made out and the outlines of the cells are seen, but the cell bodies stain very poorly and are very cloudy, while the nuclei have entirely disappeared, not even fragments being visible. Occasionally the walls of an artery on the margin may resemble a normal artery, but the vessel is blocked by a thrombosis. Beyond the area of cell-death is a narrow zone of marked infiltration with leukocytes. On the boundary of these two zones and in the area of infiltration bacteria of various kinds can usually be demonstrated by appropriate methods.

As to the nature of the pathological process various theories have been held. By some it is regarded as simply a marantic gangrene. With that idea my experience is not in harmony. Among the hundreds of cases of marasmus seen in the New York Foundling Hospital yearly, the disease is practically never seen. It occurs in children of two years or more, usually well-nourished previously, but prostrated by some severe, acute illness. Thrombosis of the bloodvessels has been held by some to be the cause of the disease, but a little study soon convinces one that the thrombosis is a secondary phenomenon, not the primary process. Woronichin, finding some slight changes in the nerves in the neighborhood of the gangrenous area advanced the theory of a causative "disturbed enervation," but the distribution, manner of extension, and all the clinical features are against that explanation. Although the specific organism has not yet been identified, there is good reason to believe that the disease is due to the invasion of bacteria in a tissue already prepared for their growth by a severe stomatitis and in an individual whose powers of resistance have been greatly lowered by acute disease (measles, whooping-cough, etc.).

**Symptomatology.**—The disease almost always begins in a severe ulcerative stomatitis. The gums have the appearances belonging to that affection. About the bases of one or more of the teeth there is the characteristic line of necrosis. Salivation is present, but not marked. The first sign of the onset of gangrene is the change in the odor of the breath and saliva. Instead of the foul odor that belongs to ulcerative stomatitis we get the horrible stench that usually accompanies a wet gangrene of any part. At this time if examination is made we find the teeth in the affected area loosened; if pulled their roots are found bathed in thin, dark fluid, which emits the characteristic odor. The periosteum is loosened about the alveolar process and there may be some superficial

FIG. 36



Noma. (Schamberg.)

necrosis of the bone. The process more often attacks the upper jaw (Fig. 36). Within twenty-four to forty-eight hours the tissues overlying the affected teeth show a deep bluish-green color underneath the skin, very much like a deep bruise. At this time, also, swelling appears and a deep induration of the part can be made out. Gradually the color of the area deepens until there is a small circle showing the characteristic greenish-black hue of gangrene. Meanwhile there is a further separation of the periosteum from the underlying maxilla, together with more superficial necrosis of the bone. We may find the process extending upon the bone well up toward the orbit, before there is much breaking down of the skin. The sloughing begins in the centre of the area, first

appearing upon the lip or cheek, and once begun extends rapidly, destroying the whole lip or cheek, the side of the nose, laying bare the bony parts beneath, and producing the most horrible sight that one is called upon to see. With the involvement of the cheek there is an almost constant flow of saliva from the corner of the mouth, bearing with it the discharge from the gangrenous area. The odor pervades the whole room or ward and is very sickening. The process may involve both sides (Fig. 37).

The general condition of these cases varies greatly. In some instances, it is said, the children do not appear to be very ill, some even sitting up in bed, apparently undisturbed, and picking out the loosened teeth

FIG. 37



Noma. (Schamberg.)

or bits of necrotic tissue. Usually, however, the patients are markedly prostrated from the beginning, the temperature is high— $102^{\circ}$  to  $104^{\circ}$  F.—and the pulse correspondingly rapid. It is remarkable that there is little complaint of pain, and the children continue to take nourishment fairly well. In my experience the disease is very soon complicated by a septic bronchopneumonia, which is evidenced by a higher temperature— $105^{\circ}$  to  $106^{\circ}$  F.—more rapid pulse and respiration, greater prostration, and, if the child lives long enough, signs of areas of consolidation, particularly in the lower and posterior parts of the lungs. Death usually occurs from exhaustion. It is not very uncommon to find an extensive diphtheria of the nasopharynx, pharynx, and possibly the larynx as a terminal complication.



**Prognosis.**—The course of the gangrené is usually rapid, terminating fatally in from one to three weeks. Instances of spontaneous recovery are recorded. In these a line of demarcation forms, the slough separates, the general condition improves, and recovery ensues, but with a horrible deformity from the destruction of the soft parts of the face.

From 70 to 90 per cent. of all cases are said to be fatal. I have never seen but one case get well, and that patient had lost one-half of the lower maxilla.

**Treatment.**—The vital point in this regard is prevention. The careful, antiseptic treatment of the mouth in all the infectious diseases of children, especially measles, is essential. The appearance of ulceration of the gums should be the signal for increased vigilance and active treatment. The ulcerated area should be scraped clean or touched with nitric acid, and every effort should be made to strengthen the child by increased feeding and alcoholic stimulants. If the necrosis of the gum spreads the loosened teeth should be removed, and the necrotic tissue, bone as well as soft parts, scraped away. It is by these methods that I believe the disease is to be arrested. When the process has once involved the soft parts the chances of successful treatment are reduced to a minimum. The application of caustics, such as nitrate of silver, chloride of zinc, nitric acid, is often advised, but they are practically useless and should be abandoned. Valuable time is lost by using them. The gangrenous area in the soft tissue should be destroyed with the actual cautery, the cauterization being carried well beyond the apparent line of gangrene. The underlying bone should be scraped thoroughly, teeth being removed, and care taken that no foci are left in the alveoli. By this method von Raube, of Berlin, has reported a number of successes, and Bainbridge has saved some cases in the New York City Children's Hospitals.

## DISEASES OF THE PHARYNX.

### ACUTE PHARYNGITIS.

Acute inflammation of the pharynx in practice includes an acute catarrhal inflammation of the pillars of the fauces, the uvula, tonsils, lateral and posterior walls of the pharynx. It is quite regularly accompanied by a similar process in the nasopharynx, which may be of more importance than the visible lesions of the pharynx. It may be preceded or followed by similar inflammation of the nose or of the larynx, trachea, and bronchi.

It is well known that acute inflammation of the pharynx occurs as an early symptom in many of the infectious diseases, especially measles, scarlet fever, diphtheria, and influenza. It may be primary, and is then most often due to exposure to cold or wet, or in our cities to exposure to high winds laden with the dust and dirt of the streets, or to digestive disorders. It may be the beginning of what is so commonly designated as a "cold," the explanation of which is probably a bacterial infection,

and then the inflammation will usually extend to both nose and throat, or it may be simply an incident of acute catarrhal processes beginning in other parts of the respiratory tract. Undoubtedly it is more common in mouth breathers, especially in those suffering from adenoids. Certain children seem to be peculiarly susceptible and have repeated attacks. These are commonly explained on the basis of rheumatism, but with very little reason. The explanation of repeated attacks of acute pharyngeal inflammation will much more often be found to be mouth breathing from obstruction in the nose or nasopharynx, improper feeding, or the persistent use of too warm baths by which resistance to exposure to cold is lowered.

**Pathology.**—The lesions are those of any acute inflammation of a mucous membrane, acute congestion and swelling, usually with some lessening of the normal mucous secretion. Later, the congestion disappears, the swelling lessens, the mucous membrane becomes relaxed, and the secretion of mucus is increased. Every case should be examined for the presence of adenoids or other cause of obstruction of the nasal passages.

**Symptomatology** —The affection generally begins in a mild way with some soreness of the throat, difficulty in swallowing or actual pain, especially if the nasopharynx is involved, and slight constitutional disturbance. Usually there is but little, if any, fever, but in some children the onset will be acute, severe, and attended with high temperature and marked prostration, exactly as if the children were beginning an acute infectious disease. Inspection shows a more or less general injection of the pharyngeal tissues, which are often dry as well as red, but may be covered with mucus. The cervical lymph nodes may be slightly swollen. If the temperature is raised there is a corresponding quickening of the pulse. The process usually subsides gradually after the first day, but may for a day or two increase in severity. The course rarely covers more than four or five days. If the process extends it is then followed by an acute laryngitis or bronchitis, which may be of more importance than the inflammation of the pharynx.

**Diagnosis.**—Inspection will reveal the condition. The important point is to be sure that one is not dealing with an acute infectious disease. Naturally, this is to be most feared in the cases with high temperature. In a family of children it is a good practice to separate a suspicious case until the question can be satisfactorily answered. Scarlet fever will promptly announce itself by the eruption. Measles may now be distinguished, in most cases at least, by the presence or absence of the Koplik spots. Cultures settle the possibility of diphtheria. With these questions disposed of, the affection is a matter of a few days' time.

**Treatment.**—The prophylaxis has been sufficiently indicated in discussing the etiology. Removal of obstructions to nasal breathing and correction of improper feeding or bathing habits are of importance. The affection is self-limited, but treatment may be of value for the patient's comfort or to prevent the extension of the process to the larynx and bronchi. The children should remain in-doors and a mild laxative



be given. For most children the milk of magnesia, 4.0-15 c.c. (5j-iv), or the effervescing citrate, 90-120 c.c. (5iij-iv), answer very well. Local applications are desirable, but young children so often resist any attempt in that direction that it may be impossible to use them. Beneficial results are secured in these cases by irrigating the pharynx through the nose with small quantities of a 2 per cent. solution of boric acid, normal salt solution, or a 1:4 solution of glycothymoline. The Birmingham douche is a very convenient device for introducing these solutions, but a blunt-tipped glass syringe or even a teaspoon will answer the purpose. Such irrigation may be repeated every two or three hours without danger to the Eustachian tubes. In all cases where irrigation is called for it is necessary to consider the infection and congestion of the Eustachian canals that may be associated with disease of the nasopharynx. Cracked ice may be given to be held in the mouth. Cold compresses applied to the neck and renewed every hour may be found useful. The diet should be liquid and should be given cold or but moderately warm. In older children any of the above solutions may be used as a gargle, but on account of the presence of inflammation in the nasopharynx it may even in these cases be desirable to introduce the fluid through the nose. If there is a temperature or much constitutional disturbance small doses of phenacetin, 0.130 gm. (2 grains), to a three-year-old child will give relief. After the acute symptoms have subsided the affection is well left to nature's resources.

#### **SIMPLE CHRONIC PHARYNGITIS (ELONGATION OF THE UVULA).**

Simple chronic inflammation of the pharynx is rarely seen in childhood, except as an attendant of chronic processes in the nose or nasopharynx. We do, however, see a chronic enlargement and elongation of the uvula which may properly be considered in this connection.

**Etiology.**—This condition of the uvula is by some regarded as congenital. More commonly it seems to be the result of repeated attacks of acute pharyngitis produced in one or the other of the ways already considered. It is often associated with chronic hypertrophy of the tonsils.

**Symptomatology.**—The most common symptom is a persistent cough, a cough which is often regarded as due to a bronchitis and treated accordingly. The cough is especially marked when the child is lying down or sleeping. In some cases the enlarged uvula may give difficulty in sucking or swallowing. Upon inspection the elongated uvula is readily seen hanging from a relaxed palate and resting upon the base of the tongue. The part is usually pale and oedematous. In older children the condition gives rise to frequent efforts at clearing the throat, "hacking," and expectoration. There may be complaint of soreness of the throat and the constant efforts at relief only aggravate the condition. As a rare condition the uvula is found congenitally enlarged and bifid.

**Treatment.**—If the affection is associated with chronic enlargement of the tonsils or the presence of adenoid vegetations the removal of these conditions may suffice to correct the condition of the uvula. In very mild cases astringent gargles, such as 0.520-0.650 gm. (8 or 10 grains) of alum to 30 c.c. (1 ounce) of water, or the application of a 2 per cent. solution of nitrate of silver, may be tried. In marked cases it is best to remove the uvula. This can be easily done by grasping the tip with a pair of long forceps and snipping the uvula above with scissors, cutting a little obliquely. Care should be taken not to remove too much of the uvula, as free hemorrhage may result. It is to be remembered that the stump may be very painful for some days or even a week after the operation.

#### CHRONIC FOLLICULAR PHARYNGITIS.

This is a condition of chronic inflammation of the small masses of lymphoid tissue normally present in the posterior wall of the pharynx. It is regularly associated with chronic enlargement of the tonsils and the presence of adenoid growths in the nasopharynx. It may be an independent condition after the removal of tonsils or adenoids or in their absence. Its etiology and pathology are essentially those of these conditions. It is seen in children of poor vitality.

**Symptomatology.**—In most cases there are no symptoms at all referable to the pharyngeal lesions, and they are discovered by accident in the course of examination of the throat. In certain cases, however, especially after exposure to cold during the winter months, these little growths become somewhat swollen and congested, and may then give rise to discomfort, a sense of rawness or even pain in the throat, and hawking to clear the throat. The appearances are characteristic. The little rounded growths, pale or reddish in color, are seen projecting slightly above the surface of the pharyngeal wall, and scattered at intervals over it. They very much resemble the corresponding lymphoid masses at the base of the tongue.

**Treatment.**—If adenoids or enlarged tonsils are present the removal of these may be all that is required. The pharyngeal growths rarely demand removal. If it be necessary this is best accomplished by burning with the galvanocautery. In the absence of this the growths may be cauterized by crystals of chromic acid fused on a glass rod. Only two or three points should be touched at one sitting and a number of applications may be required.

#### AFFECTIONS OF THE UVULA.

The uvula is commonly considered simply as one of the structures involved in pathological processes of the throat and receives no special mention. It does, however, possess a certain individuality. It may be

congenitally short or absent, bifid or abnormally long. The latter condition may be a factor of importance in the production of chronic cough or even asthma.

In acute inflammation of the throat the uvula often suffers to a striking extent; the swelling and tenderness of this part not infrequently constituting an important element in a "sore throat." Huber, of New York, has reported an instance illustrating the fact that the uvula alone may be involved in the inflammation. An infant ten months old was apparently well until two hours before it was seen. It then developed a constant irritating cough, accompanied by considerable gagging. A little later a prominent red mass was observed in the mouth. There were paroxysms of coughing which interfered with both nursing and deglutition. The general symptoms were alarming and the child was in considerable distress. On examination the mass in the mouth was found to be the elongated and inflamed uvula, measuring an inch in length and half as much in width. It was red and edematous, but the throat was otherwise normal. The symptoms were relieved by multiple needle punctures and the use of ice, both internally and externally. Chronic hypertrophy of the uvula has already been considered under Chronic Pharyngitis. Nevus of the uvula is sometimes seen and papilloma may occur.

## CHAPTER X.

### DISEASES OF THE STOMACH.

#### THE DIGESTIVE ORGANS AND DIGESTION IN INFANCY AND CHILDHOOD.

BOTH in their structure and functions the digestive organs of infants present certain peculiarities which serve to explain to some extent the qualitative as well as quantitative differences that undoubtedly exist between infantile and adult digestion. The more important of these it is advisable to consider as a preliminary to the study of pathological conditions affecting these organs.

The salivary glands are present at birth, and are functionally active at this time. The digestive power of their secretion has, however, long been questioned or even denied. Although previous observers had detected ptyalin in the saliva of newborn infants, Zweifel's findings that ptyalin was present in the parotid glands at birth, but not in the submaxillaries until after the second month have long stood as authoritative. Shilling in 1903 proved, however, that ptyalin could be found in the submaxillaries of infants from nine days to six weeks old. Shaw, of Albany, has recently published a series of experiments which prove that the saliva of infants possesses some diastatic power even from birth. The investigations of Chittenden have also shown that ptyalin is not, as previously supposed, at once rendered inert by mixture with the acid contents of the stomach, but that its action continues until the free hydrochloric acid reaches one-tenth of 1 per cent. While, therefore, it must be granted that the saliva of infants is but a feeble digestive agent, it does possess some power, a power which partly explains the practical experience that even the youngest infants can at times take cereal decoctions with advantage. The digestive power of the saliva rapidly increases during infancy. Korowin was unable to find any difference in power between the saliva of a healthy adult and that of an eleven-months-old baby.

**The Stomach.**—At birth the stomach is very small, its cubic capacity averaging 1 ounce, and it often appears as if simply a dilated portion of the intestinal canal rather than a distinct organ. Its rate of growth is, however, very rapid. At three months its average capacity is four and a half ounces, at six months six ounces, and at a year nine ounces. The walls at first are thin and especially lacking in muscle tissue, but in this respect also growth is rapid. It is often said that in early infancy the position of the stomach is vertical, and that the organ only gradually assumes the horizontal position characteristic of later life.



To a certain extent such statements are misleading. The stomach in infancy occupies a somewhat more upright position than in later life, chiefly owing to the fact that the fundus is, as yet, but very little developed, but it is never vertical, and by the end of the first year the position is practically that normal in later years. Microscopically, according to Baginsky, the differentiation of the cells of the mucous membrane into several types can be recognized in the newborn.

The gastric secretion of infants in health is a rather thick, colorless, tenacious, mucous material which is usually strongly acid in reaction, but sometimes neutral. As a rule, it contains free hydrochloric acid, but not always, and pepsin or pepsinogen. Its most constant and characteristic constituent, however, is the so-called lab-ferment, which is found both in sick and in well children and at all stages of digestion. It is to this lab-ferment that the prompt coagulation or clotting of milk of any kind which takes place upon its introduction into the stomach is due. This clotting takes place no matter what the reaction of the stomach contents and entirely independent of the presence or absence of free hydrochloric acid. Whether the hydrochloric acid itself exercises an independent influence upon this process of clotting is not known. It is, therefore, to the presence of this lab-ferment that the most striking difference in the digestion of human and cows' milk—the well-known fine clotting of the one in contrast to the thick, tough, almost glutinous clots formed by the other—must be ascribed.

To the hydrochloric acid is ascribed the chief role in the gastric digestion of the infant (Unger). The secretion of this acid begins with the reception of food and continues throughout the digestive process, yet it is often impossible to demonstrate the presence of free hydrochloric acid in the infant's stomach until near the end of the digestive process, one and one-quarter to two hours after feeding, the reason for this being that the acid as it is secreted is taken up and chemically fixed by the albumins and salts of the food, and only when the affinities of these constituents of the food are satisfied does free acid appear in the gastric contents. The proportion of acid found in the infant's stomach is always much lower than that of the adult organ—0.13 per cent. as against 1.5 per cent. to 3.2 per cent. (Læo).

It has been demonstrated in Heubner's clinic that milks of various kinds differ in their ability to take up hydrochloric acid according to their content of albumin and salts. Cows' milk is said to take up most acid, mares' milk less, and human milk least of all, only from one-third to one-half the amount taken up by cows' milk (Miller), another fact which helps to explain the differences in digestibility.

In infants fed entirely upon milk, lactic acid is a constant constituent of the gastric contents, this acid having its origin in the milk-sugar. That lactic acid probably exercises some influence upon digestion is now known; as is shown by the easily digested mother's milk, where the amount of milk-sugar is conducive to lactic acid formation.

A question of considerable importance, in infantile digestion, especially with relation to the diagnosis of conditions of pyloric obstruction,



is that of the duration of the stay of food in the stomach. That the contents of the stomach pass very rapidly into the duodenum and that the stomach may be empty within one-half hour of a nursing in a young infant, is well known. Epstein gives one and one-half hours as the maximum time for the evacuation of the stomach in a breast-fed child. Naturally, as the size of the stomach increases and larger quantities of food are taken, the food remains longer in the stomach. In artificially fed children, especially in those taking cows' milk, there is an appreciable prolongation of this period. Even in the intervals between the periods of digestive activity the stomach is not entirely empty. In the resting organ a small quantity of yellowish fluid will be found, which contains all the constituents of the gastric secretion in concentrated form and gives the biuret reaction (Unger).

Concerning the extent of the digestive process which is carried on in the stomach there has been considerable discordant investigation. It is now generally agreed that only a partial peptonization of the milk occurs in the stomach. It is usually accepted that this peptonization is accomplished by the agency of the hydrochloric acid and pepsin, but some hold that the lab-ferment exercises a distinct power in this direction, as peptone can be found in the stomach contents before there is sufficient acid and pepsin to explain their presence. However that may be, we are satisfied that the function of the stomach is largely that of a reservoir and that the greater part of digestion is conducted in the small intestine. Here the acid gastric contents are subjected to the combined action of the bile, the pancreatic juice, and the intestinal secretion. It is generally accepted that bile is relatively more abundant and more effective in the infant than in adults, the relatively larger liver being assumed to produce a larger quantity of bile. There are no differences, so far as known, in the action of the bile in infancy and in later life. The pancreatic secretion shows both trypsin and steapsin at birth, but the amylopsin has been said not to appear until the second month of life. Moro has, however, recently established the fact that it is present in the newly born. Of the powers of the intestinal secretion in infancy, practically nothing is known. In the intestine, however, the partially peptonized proteids are rendered soluble through the action of the trypsin especially, and prepared for absorption. The fats taken in are split by the pancreatic juice into fatty acids and glycerin, and these acids are then saponified by the action of the bile, the result being a fine emulsion of fat which is readily absorbed. Sugars are, of course, absorbed in their natural condition. Starch, if present, is affected, to some extent at least, by the saliva, and it now appears is subjected to further digestion by the pancreatic secretions. Clinically, it has been established beyond doubt that infants, even in the first months, may take and digest starch in small amounts. Absorption for the most part is carried on by the small intestine, to a much less degree by the large intestine. For this reason disturbances of the small intestine produce diarrhea, with frequent watery passages, followed by rapid wasting. In affections of the colon alone the diarrhea is less watery and the wasting is much less

# PLATE V

Fig. 1

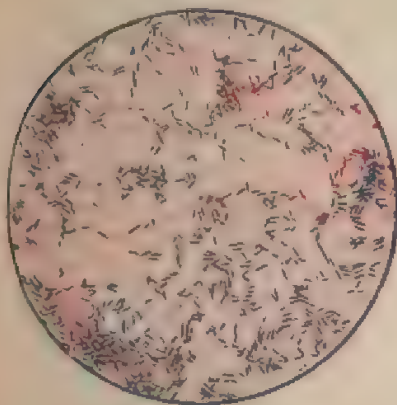


Fig. 2

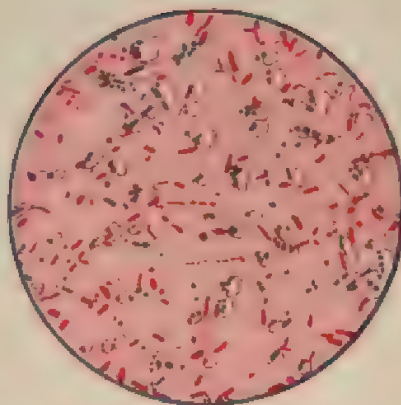


Fig. 3

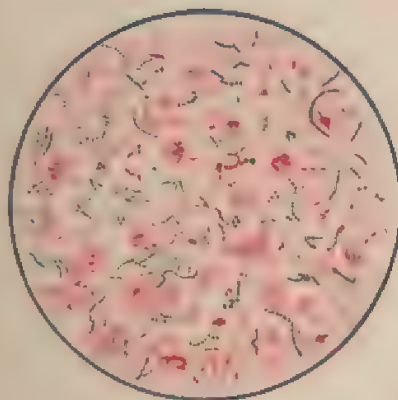


Fig. 4



Fig. 5

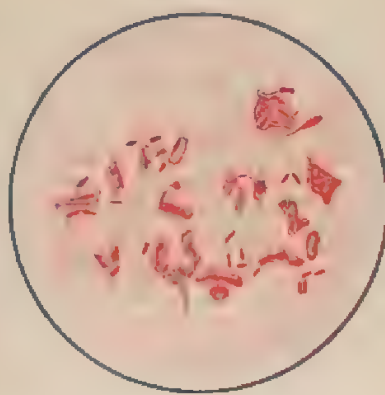


Fig. 1. Normal stool of breast-fed infant. Smear.  $\times 1000$   
 Fig. 2. Normal stool of bottle-fed infant.  
 Fig. 3. Mucus from an infant's stool, in streptococcus enteritis. Stained  
 with carmalum. Each micrograph is a detail. (After Hirsch.)  
 Fig. 4. Dysentery bacilli. Smear from a small clump of pus. Stained with dilute  
 carmalum.  $\times 1000$   
 Fig. 5. Furthest portion of an infant's stool in a case of acute colitis. Details as  
 in Fig. 1.



rapid. When the intestinal contents are delayed in their transit through the canal, absorption goes on to such an extent that the feces are reduced to dry, hard masses, which are moulded into scybala by the peristaltic action of the intestines.

**The Feces.**—The normal breast-fed child has from one to five movements daily, which are at first unformed, thin, bright yellow, smooth and with little odor. (See Plate V.) After the first few weeks the passages show some consistency, but otherwise remain the same. The artificially fed child taking cows' milk, usually has less frequent movements, which are somewhat formed, lighter yellow in color, not so smooth, and having a more or less pronounced and rather offensive odor. In either case the first evidence of intestinal disturbance is an increased number of movements which are first of all looser and then show a change of color, becoming green. Later, the movements show curds, which are usually the undigested proteid, and, perhaps, mucus or blood. Pus is not often visible in the movements of children. The curds are usually small, white masses, closely resembling those seen in sour milk, but fat also may present itself in masses, which are, however, light yellow in color, more translucent and oily in appearance. The fatty masses are readily soluble in alcohol or ether. These masses of fat may come from the food or from cod-liver oil or other fat administered medicinally. The greenish color so characteristic of the diarrheal movements of children is said to be due to a change in the reaction of some part of the intestinal canal, it becoming alkaline instead of acid—for in the normal condition the tract is said to have an acid reaction throughout. Bacterial action is doubtless concerned in the change. It is to be remembered that quite normal stools may, upon exposure to the air, shortly show a greenish tinge upon the surface.

#### RECURRENT VOMITING.

Under this title is indicated an affection called also Periodic or Cyclic Vomiting, first described by Gee, characterized by more or less frequent attacks of severe vomiting, with or without fever, and accompanied by marked prostration, and not to be accounted for by indiscretions of diet, organic disease, or other common cause of such disorders.

**Etiology.**—The affection is not usually met with in infancy, but belongs especially to children from six to twelve years of age. A number of cases, have, however, been met with in children not over three years old. Girls are said to be attacked more often than boys. A gouty, rheumatic, or neurotic family history is found in most of the cases. For a long time the exact nature of the affection has been the subject of much discussion, the weight of opinion inclining to the view that the disorder was not dependent upon the organic disease of the alimentary tract, or, indeed, of any organ, but was rather a gastric neurosis. Certain observations have shown that during the attacks there is a marked diminution in the excretion of uric acid, the ratio of uric acid to urea

present in the urine rising from a normal of 1 to 40 or 50 to 1 to 80, or at times even as high as 1 to 150. These observations had served to connect the disorder with the group of disturbances attributed to that very vague and unsatisfactory condition known as the uric acid diathesis.

It was believed that the underlying condition was a disturbance of metabolism resulting in a diminished excretion of uric acid, and that these attacks of vomiting were exactly similar to the attacks of migraine from which certain individuals suffer. Recently, attention has been called to the fact that in most of these cases, if not all, the attacks are accompanied by the presence in the urine of those products of imperfect metabolism, acetone, diacetic acid, and  $\beta$ -oxybutyric acid which have been found in the urine in cases of diabetes. The original observation of Marfan has been confirmed in this country by the work of Edsall, Pierson and others. The exact significance of these observations with relation to the underlying process is not yet clear. It may be that the real cause of the disturbance is error in the internal metabolism of the tissues similar to that present in diabetes. Edsall inclines to the view that the primary disturbance is one of digestion, and that careful observation will show some irregularities of digestion preceding the attacks. For the present we can, therefore, only say that most of these cases show the condition known as an acid intoxication dependent either upon an error of digestion or of the internal metabolism. It is not at all probable that all cases of this disorder can be accounted for upon this one basis, but that careful study will show some other cause or causes for at least a minor portion of the cases. The affection is not a frequent one and in any suspected case care must be exercised not to overlook organic lesions or other definite cause of the attacks.

**Symptomatology.**—The onset of the attacks is usually sudden, and is not accounted for by dietary indiscretions. It was formerly asserted that there were no prodromata, but more careful observation has shown that the children do, for at least a day or two before each attack, show some indisposition. This may be limited to an appearance of peevishness or listlessness with dark lines under the eyes and loss of appetite. In other cases there is a slight fever— $100^{\circ}$  to  $101^{\circ}$  F.—or the movements of the bowels become notably pale or white. Looseness of the bowels may be associated with the onset, but the opposite condition is more frequent. Undoubtedly in some cases the attacks occur in children in apparently perfect health.

The vomiting once begun is frequent and persistent. Pierson reports the case of a child who in one attack vomited eighty-seven times in forty-four hours and in another fifty-two times in thirty-seven hours. The stomach is intolerant of anything and every attempt at medication or feeding by that route results in a repetition of the vomiting. All the natural consequences of such a disturbance follow in due course. The eyes become sunken, the tongue is coated, the skin dry, the abdomen is retracted, the urine is scanty and high-colored and has a characteristic sweetish odor, weight is lost rapidly and the prostration becomes extreme and the bowels are usually constipated. The temperature usually rises



and may reach 103° or even 104° F. during the attack. Each attack lasts for two or three days and is regularly followed by a gradual return to normal. It may be weeks before the child recovers from the prostration of an attack.

The periodicity of the attacks is very irregular. They usually recur at intervals of weeks or months. Until the observations upon the presence of acetone and its congeners in the urine were made there was no clear explanation of the recurrence. It now seems that by watching the urine for the appearance of these bodies the advent of an attack can be foretold and provided for or even prevented. In most of the cases heretofore reported the persistence of the affection has been marked.

**Diagnosis.**—In a first attack this may present some difficulties. The natural supposition at first sight would be that the child was suffering from having eaten some undigestible food, but this can be readily excluded by the history of the case, and the course of the affection, the complete intolerance of the stomach for a period of several days followed by prompt recovery being quite characteristic. In infants pyloric stenosis should not be overlooked as a cause. Meningitis can be excluded by the absence of any of the focal symptoms of that affection. The urine should be carefully watched to exclude the possibility of nephritis, and, in the light of our present knowledge, tested for the presence of acetone.

**TESTS FOR ACETONE.** *Legal's Test.*—This test may be applied to the freshly voided urine, but it is much better to first distill the urine. The test solution is made by dissolving a few crystals (0.015-0.020 gm. = 2 or 3 grains) of sodium nitroprusside in a few cubic centimetres of water, to which are added a few drops of a 40 per cent. solution of sodium hydrate. When 2 or 3 c.c. of urine are added to such a solution a red color develops, which, in the presence of acetone, is replaced by a purple or violet-red color on the addition of a few drops of acetic acid.

*Lachen's Test.*—This and the following (Reynolds') are both applicable only to the distilled urine. A few cubic centimetres of the distillate are treated with several drops of a dilute solution of iodopotassic iodide and sodium hydrate (easiest made by adding a few drops of the 40 per cent. or other strong sodium hydrate solution to a dilute Gram's solution), when in the presence of acetone a precipitation of iodoform in crystals occurs, which at once is recognizable by the yellow color and characteristic odor.

*Reynolds' Test.*—A few cubic centimetres of the distillate are treated with a small amount of freshly precipitated yellow oxide of mercury. This is prepared by precipitating a solution of bichloride of mercury with an alcoholic solution of sodium hydrate. If acetone be present a black color, due to the formation of sulphide of mercury, will result in the clear filtrate upon the addition of a few drops of ammonium sulphide. In making the above tests it is always best to use freshly prepared solutions, but inasmuch as the reactions are qualitative only, it is not necessary to have mathematically accurate solutions.

It is to be remembered that acetone is found in the urine in a number of other conditions, notably in diabetes, where its occurrence in considerable quantities in association with sugar may be said to be diagnostic, and when also the amount of acetone is in proportion to the severity of the disease. Acetone has also been observed in the urine in typhoid, pneumonia, scarlatina, measles, acute miliary tuberculosis, acute articular rheumatism, and septicemia.

**Prognosis.**—So far as the individual attack is concerned the prognosis is generally good. Griffith has, however, seen two cases result fatally, and the extreme exhaustion of the little patients makes the affection a grave one in most cases. As above stated the attacks tend to recur with marked persistency and there seems little tendency to cure without proper treatment. The patients may, however, maintain their nutrition well and otherwise appear to enjoy good health.

**Treatment.**—The attacks themselves are self-limited and for evident reasons treatment after an attack is once established is difficult and not likely to be very effective. Evidently the desirable thing is to get at the underlying condition and prevent the recurrence of the attacks. In the light of our present knowledge of these acid intoxications the control of the diet is of the utmost importance. As it is apparently the carbohydrate digestion or metabolism that is at fault, foods of this class must be reduced to a minimum. Fats also are said to be poorly borne except in the form of fresh butter and we are therefore reduced to a dietary consisting in great part of nitrogenous foods with green vegetables and stale bread or rusks. Exactly as in diabetes it is rarely of advantage to entirely forbid the use of carbohydrate foods, so in this disorder they should not be entirely excluded, but limited to small quantities.

Working on these lines a dietary for a child six years of age might be constructed as follows:

**Breakfast.**—Eight to twelve ounces of fresh milk; one or two tablespoonfuls of a wheaten cereal with milk; dry toast or zwieback or rusk with butter; a soft-boiled egg, or occasionally fresh fish.

**Dinner.**—Broth or soup (clear); a chop, bit of steak or roast beef, without fat; spinach, celery (stewed), string-beans, or green peas; stale bread and butter; milk, if desired.

**Supper.**—Eight or twelve ounces of milk; a bit of chicken, or fish (boiled), or occasionally an egg (boiled); dry toast, or rusk, or zwieback, and butter.

Sugar and sweets should be rigidly excluded.

Additions to the dietary should be made with care. Dieting on such lines will serve to greatly reduce the frequency of the attacks. Since the discovery of the condition of acid intoxication in these cases the continuous administration of sodium bicarbonate in quantities sufficient to neutralize the urine, 0.65 gm. (10 grains) or more, three times a day, has been found extremely useful. By these means the recurrence of the attacks has been completely interrupted in a number of cases. The general hygiene of the patient must be cared for.

If, in spite of such measures or in their absence, an attack seems imminent, but is not yet fully developed, the administration of sodium bicarbonate in full doses as much as 8.0 gm. (2 drachms) being given in divided doses during a day, seems capable of mitigating the severity of an attack, or even checking it. Such large doses of the alkali should, naturally, not be continued for many days, but may be safely persisted in until the urine is quite alkaline, and the attack is over.

With a rheumatic record the use of soda salicylate is advisable. In all cases water should be given freely and the bowels kept open.

In case the patient is already vomiting severely, it will be best to stop all attempt at medication or feeding by the mouth, remembering that the attacks usually terminate at the end of two or three days. Ice may be allowed, to suck, or water given in teaspoonful doses, if it does not bring on vomiting. When several hours have passed without vomiting the administration of food may be begun in similar minute quantities, whey or broths being preferable to milk. A teaspoonful may be given every fifteen or thirty minutes in the beginning, the quantities and intervals being both gradually increased. Later, kumyss, matzoon, equal parts of milk and lime-water or milk and Vichy water may be used. During the height of an attack the sodium bicarbonate or other necessary medication may be given by the rectum, but the stomach had best be spared the administration of any medicine. In desperate cases morphine may be given hypodermically, but only to tide over an emergency. In the light of our present knowledge the subcutaneous or intravenous administration of a feeble solution of sodium bicarbonate might be of help in a critical case, but so far as known this has not yet been resorted to. From what we have seen of the use of such solutions in diabetic coma, we know that this treatment can be safely employed in conditions requiring it, but any such painful treatment is a much more serious undertaking in a child than in an adult.

#### GASTRALGIA.

The term gastralgia may be applied to any pain in or referred to the stomach. Practically, however, we employ it to cover attacks of pain referred to the stomach and not accountable for by definite lesion or disturbance either of the stomach or other viscera. The clinical conception is, therefore, of a sensory neurosis of the gastric nerves—a neuralgia. The etiology of such a nerve disturbance must be that of a neuralgia affecting any other nerve of the body, but referred to the gastric nerves for reasons quite beyond our present knowledge. It may, therefore, be conceivably produced by a condition of nervous exhaustion, by a gouty or rheumatic disposition, by anemia, or by whatever other influences may impair the nutrition or function of the nerves. The more carefully we try to understand just what is meant by a neuralgia of the gastric nerves the more restricted will become the field for the application of the term.



**Pathology.**—Of the changes in the nerves in gastralgia we at present know nothing. The condition in the other parts of the body would be that of some one of the general conditions above suggested.

**Symptomatology.**—In a true gastralgia the pain comes in attacks of greater or less severity and lasts for a varying length of time, a few minutes or hours. The onset of the pain is sudden, usually severe, and the patient may be quite prostrated by the attack, but recovers promptly upon the remission of the pain. The attacks of pain have no relation to the taking of food, and pressure upon the epigastrium may be grateful rather than distressing. If the pain is severe vomiting may be excited, but this is generally absent. There is no disturbance of pulse, respiration, or temperature, and the general health of the individual may be good.

**Diagnosis.**—This is the essential point in reference to this affection. The common error is to loosely speak of a gastralgia, when more careful observation would show that the supposed neuralgia is to be satisfactorily explained by some lesion of the stomach or neighboring organs. It is a familiar fact that young children will, when asked "Where is the pain?" promptly lay the hand upon the epigastrium, when the physical examination shows that the lesion is in the lung, the pleura, the heart, the appendix, or even the spine, and in those of more advanced years the seat of pain may not be a safe guide as to the location of the disease. Any complaint of persistent or recurrent pain in the gastric region should call for a careful study of the case and a thorough physical examination. The history of the case and the absence of any marked disturbance of the temperature, pulse, or respiration should enable us at once to exclude all acute inflammatory or suppurative conditions of the stomach or neighboring viscera. We should, then, consider the possibility of the presence of some chronic disorder of the gastroenteric tract, of which we should expect evidence in the condition of the breath and tongue, disturbance of the appetite, vomiting, constipation or diarrhea, and the like.

A careful physical examination should then exclude the presence of any disorder of the heart, lungs, liver, or spleen which might account for the pain. Enlargement of the spleen from malaria or other cause is one of the rarer causes of epigastric pain in children. The urine should be examined to exclude disease of the kidney and the position of those organs taken into account. Finally, the spine of the child should be carefully examined for deformity, rigidity, or other sign of beginning disease of the vertebræ. The orthopedic surgeon is familiar with cases of Pott's disease that have been treated for weeks or even months for gastralgia or indigestion, when an examination of the spine would at once have disclosed the seat of real trouble and rendered proper treatment possible at the time when it is of the utmost importance. Only when we have thus gone over a case thoroughly and excluded every other possible cause for the pain may we safely speak of a gastralgia in a child.

**Treatment.**—For the attacks of pain relief may be secured by putting the patient to bed and applying a hot-water bag to the epigastrium.

More effective still is the application of a mustard paste or turpentine stupes to the epigastrium. The former is much easier of application. Internally 0.60-2.0 c.c. (about 10 to 30 drops) of brandy or gin in hot water or a few drops of spirits of chloroform or camphor in a teaspoonful of cold water will be effective. The more important problem in chronic cases is the prevention of the attacks. If we can get at a definite cause for the complaint, rheumatic or gouty diathesis, anemia or the like, the proper line of treatment should be followed. In the absence of such indication the diet should be carefully regulated to the end of improving nutrition, overexercise forbidden, and adequate rest secured. The systematic use of Fowler's solution, begun with 0.065 c.c. (1 drop) given well diluted in water and increased gradually to the limit of tolerance, will be found of advantage. It is much better to give the arsenic in this way than in a complex prescription, for increases can be made more readily and a larger amount will be borne without disturbance. In persistent cases an out-of-door life in the country may be effective where other remedies have failed.

#### ACUTE GASTRIC INDIGESTION.

The line between the condition designated as acute gastric indigestion and an acute gastritis is purely theoretical, yet for practical purposes it seems advisable to describe the affections separately.

**Etiology.**—An attack of acute gastric indigestion may be brought on at any time when an unusual tax is put upon the stomach. This may arise either from errors in the quantity or quality of the food taken or from other conditions which have indirectly lowered the functional activity of the stomach and, perhaps, rendered it unequal to demands which it had previously been meeting perfectly well. In infants such attacks are commonly brought on either by feeding too much at one time, or by sudden changes in the feeding, such as weaning, or substituting one food for another, especially if the new food be cows' milk, giving solid food too early, etc. Some infants show such a susceptibility to cows' milk that the giving of even a single spoonful may be sufficient to bring on a violent attack of gastric indigestion. Fortunately, such cases are rare.

In children the errors most often lie in overindulgence in pastry, candies and the like, or too hurried eating. Decayed teeth may be a cause by making mastication painful. Unless carefully watched and trained many children habitually eat too rapidly, the food is consequently imperfectly masticated, digestion is rendered more difficult and upon very slight occasion may be entirely arrested.

Of the influences which bring on indigestion through impairment of the stomach functions the most important are dentition, exposure to unusual cold or heat, violent exercise immediately after eating, or great nervous excitement.

**Pathology.**—Of this we can naturally know nothing directly, but it may reasonably be assumed that in this condition there is a sudden



arrest of the functions of the stomach, both as to secretion and motion. The normal gastric juice and the peristaltic action both fail.

**Symptomatology.**—An attack of acute gastric indigestion is most often inaugurated by more or less abdominal discomfort, associated with nausea, and followed by vomiting. The appetite is lost and the tongue coated. The pain may be severe. The vomiting is usually violent and is prolonged for some hours, but is over after a period of sleep. The vomitus shows that undigested food has been present in the stomach many hours after the normal length of time, the retention being due to the failure of the motile power of the stomach. The general symptoms which are attendant upon these attacks of indigestion are important. In the milder cases there may be none. Often there is more or less temperature—100° to 102° F.—it may be 104° or 105° F., a rapid pulse, and marked prostration. The nervous symptoms may be marked or even alarming. In some instances the child is listless, stupid, the pupils contracted, the condition suggesting opium poisoning. In other cases the child is restless, excited, and convulsions may occur. The bowels are usually constipated, but this soon gives way to a diarrhea with the passage of much undigested food. When the stomach has been well emptied the disturbance gradually subsides. The temperature and pulse fall, the mental condition becomes more natural and recovery is usually prompt. There is a tendency to nausea and vomiting, however, for some days thereafter. These attacks are usually not serious except in feeble infants, in whom such a disturbance may well prove fatal. In the summer season every such attack is of great importance, because it opens the way to more serious disturbances of the digestive organs.

**Diagnosis.**—This is not difficult, as a rule. These gastric disturbances are among the common phenomena of infancy and childhood. The history of the case usually points clearly to the nature of the affection, and the symptoms are straightforward. One may not, however, be able to say in the beginning whether the disturbance is a simple indigestion or a gastritis, nor can he be sure that the symptoms are not those that mark the onset of some acute infectious disease. Time will be required to clear up the latter question.

**Prognosis.**—This is almost always good except in the case of weak infants, to whom such an attack may be fatal, especially if the nervous symptoms are severe and convulsions occur.

**Treatment.**—The natural course of the disease indicates the proper treatment, emptying the stomach and rest for that organ. In infants this can best be accomplished in the manner in which stomach washing is done in an adult. For a stomach tube one uses a large-sized rubber male catheter, size 16 American or 24 French. This is joined by a bit of glass tubing (to allow inspection of the movement and character of the fluid passing) to about two feet of small rubber tubing connected with a glass or hard-rubber funnel capable of holding 125-175 c.c. (4-6 oz.). The child should be carefully wrapped in a sheet, with the arms at the sides, so as to prevent it from grasping the tube, and then held face upward on the nurse's lap. The tube can be easily passed through the

mouth and pharynx into the esophagus (Fig. 38). The tube should be passed over the laryngeal region as rapidly as possible to avoid gagging. Except in a comatose child it is impossible to pass the tube into the larynx. It is well to measure the distance from the tip of the *ensiform* cartilage to the chin, beforehand, as a guide to the length of

FIG. 38



Method of washing out the stomach. Note the manner of holding the child and the elevation of the funnel.

tube to be introduced, although there is no danger of passing the tube too far. Once the tube is in the stomach the funnel is raised to allow the escape of gas, then lowered to siphon out the contents of the stomach. If the child is quiet, nothing is likely to run at first, but if a few ounces of water are run in to fill the tube and start the siphon action, the

stomach will be promptly emptied. Water should then be used until the stomach washings are clear. If the siphon will not work, we may be sure that the tube has been blocked by some solid food sticking in the eye of the catheter. In that case the tube must be withdrawn, cleared, and replaced, although sometimes running in a little more water may suffice to dislodge the obstruction. Plain water at a temperature of 100° F. may be used, or normal salt solution, or sodium bicarbonate solution, 4 gm. to 500.0 c.c. (1 drachm to the pint) may be employed. Many authors recommend boric acid in the proportion of 1 : 200, or even resorcin 1 : 5000; but as cleansing is the important point, and this is accomplished best by the alkaline solution, the use of antiseptics has nothing to recommend it. It is well to use a warm solution. Collapse can be produced or augmented by cold solutions.

In children over two years of age the stomach tube cannot ordinarily be employed, because of their struggling and their ability to bite. We must then content ourselves with giving them large draughts of water with 4.0 to 8.0 c.c. (1 to 2 dr.) of the syrup of ipecac, to excite active vomiting. This is not so satisfactory as the stomach washing, but we have to be content with it.

The stomach having been well emptied, nothing but water should be given for several hours. If the attack occurs in the afternoon or evening it is best to let the child go until morning before attempting to feed it. Feeding should be resumed very carefully. In nursing infants it is best to allow the child to nurse only two or three minutes at first, prolonging the nursing time according to the indications. For artificially fed children we may use whey, albumen-water, or a weak preparation of one of the cereal foods, allowing only 15-30 c.c. ( $\frac{1}{2}$  to 1 ounce) an hour at first, gradually lengthening the interval and increasing the quantity. In the severer cases it may be advisable to begin with teaspoonful feedings. Milk should be withheld from these children for several days, and when it is resumed it should at first be given much more dilute than the child had been taking it before the disturbance, barley-water or lime-water being used as the diluent. It is very easy to bring on a relapse in these cases by too rapid progress in the feeding. With care the ordinary feeding may be resumed by the end of a week. If there are loose or decayed teeth they should receive attention.

Drugs are usually not required in this condition. If the bowels have not moved of themselves calomel may be given for that purpose, 0.0065-0.013 gm. ( $\frac{1}{16}$  grain to  $\frac{1}{8}$  grain) hourly until a grain has been taken. If the vomiting persists after the washing the following powder may be given with advantage:

R—Bismuth. subnitratls,  
Ceruleum oxalate,  
Sodium bicarbonatis . . . . . 4d 2.0 gm. (5m).  
M. et div. in chart. No. xlii.  
Sig.—(One powder to be given with each feeding.

The powder may be given dry on the tongue and washed down with a little water or milk, or it may be given in a small portion of the feeding.



Quiet and careful dieting are the essentials in the management of these cases and medication is of distinctly secondary importance. During convalescence the bowels may be constipated. A simple enema of 500 c.c. (1 pint) of water is the best means of moving them, but many children resist the administration of enemata to such an extent that it is necessary to resort to medication by the mouth. Calomel may be given again in the manner already described, or 7.50 c.c. (about a dessertspoonful) of the milk of magnesia, or 120-180 c.c. (about a glass) of the effervescent citrate of magnesia may be employed.

### ACUTE GASTRITIS.

**Etiology.**—An acute catarrhal inflammation of the stomach is relatively rare as an independent lesion, but is common enough as an accompaniment or as part of a general inflammation of the intestinal tract. It is frequently associated with the inflammation of the intestine and colon, which will be described later. It is present in many of the acute infectious diseases.

The primary or independent form may be produced by any of the causes already given for acute gastric indigestion. Whether we shall get in a given case an attack of indigestion or an active inflammation of the stomach depends upon the resistance of the stomach in that particular case and the virulence of the exciting cause. The most common cause in infants is improper feeding, especially in the case of artificially fed children. Breast milk may be so indigestible that its use produces an acute catarrh of the stomach, but such cases are very rare. In the artificially fed, acute catarrh of the stomach is common, especially upon sudden changes or some egregious error in the feeding. During the summer it is not uncommon to see this disorder in children from a single feeding of milk that has undergone change from bacterial action.

For convenience the acute gastritis excited by the administration or accidental taking of caustic poisons, such as carbolic acid, strong acids or alkalis, etc., is regularly considered under this head.

**Pathology.**—The gross changes are not marked. The stomach is found either contracted or dilated. Externally it is normal. On opening, the contents are found to consist of mucus and more or less food. The mucus is thick and ropy, as a rule, and is often quite firmly adherent to the mucous membrane. Not infrequently the mucus is mixed with more or less coffee-ground material, which analysis proves to be blood, doubtless from capillary hemorrhages, for no gross lesions of the blood-vessels can be found. The mucous membrane is swollen and more or less congested, especially along the greater curvature and near the pylorus. There may be minute hemorrhages into its substance. Microscopically there may be a loss of the superficial epithelium and some round-cell infiltration of the mucosa and, in severe cases, of the submucosa. Minute extravasations of blood may also be found. The

changes are very likely to be in scattered areas, not general. The muscular and peritoneal coats are normal.

A follicular inflammation of the stomach is a rare finding in these cases. When it is present the solitary follicles of the stomach, which are scattered at rather wide intervals through the mucous membrane, are swollen and in the centre of each follicle there is a slight superficial loss of epithelium giving the appearance of a minute ulcer, about the size of a pinhead. Rarely does the ulceration appear more considerable. These changes may be associated with those of a catarrhal inflammation. The lesion is of exactly similar type to that seen in follicular inflammation of the colon, but the follicles are not so numerous in the stomach.

A membranous inflammation of the stomach is a very rare finding in the postmortem-room. It is usually seen in association with some one of the infectious diseases. It is more often not diphtheritic. I have seen one case in which the diphtheria bacilli were obtained both in smears and culture from the membrane. It is a curious fact that in these cases of diphtheritic inflammation of the stomach the esophagus is not involved. The lesions in these membranous cases are those of a croupous inflammation of any mucous membrane. The surface of the membrane is coated with an exudate of fibrin, leukocytes, epithelium, and bacteria. The underlying mucous membrane is rough, granular, congested, and shows, on microscopic examination, a more extensive infiltration with leukocytes extending into the submucosa. There may also be small extravasations of blood. The muscular and peritoneal coats are regularly normal. A gangrenous inflammation of the stomach I have seen only once, then in association with cancrum oris. The mucous membrane of the stomach was greatly swollen and thickened by infiltration; the crests of the rugæ were coated with a croupous exudate, while the whole mucous membrane was soft, greenish black in color, and emitted the characteristic odor.

In cases of caustic poisoning the mucous membrane presents the appearance of an acute inflammation with more or less scattered ulceration, the extent of the ulceration depending upon the amount of caustic which has reached the stomach. This is usually small. The ulceration may, in rare cases, be deep enough to penetrate the walls of the stomach.

The condition of *gastromalacia* is occasionally seen in autopsies on children. This is a softening of the wall of the stomach produced by a process of self-digestion. A considerable area of the wall is reduced to a soft, gelatinous mass which readily yields to any tension or may have already permitted the escape of the gastric contents into the peritoneum. The area involved is always on the greater curvature and in its most dependent part. There are none of the usual evidences of inflammation about the margins of the softened area, or, indeed, in other parts of the stomach. Considerable importance was at one time attached to this condition, but we have learned that it has no relation to disease of the stomach during life.

**Symptomatology.**—As has already been pointed out the onset of acute catarrhal inflammation of the stomach is exactly the same as that of



acute gastric indigestion. The two affections differ only in their course. In acute catarrh the vomiting is more persistent. The vomitus contains more mucus, after a time it may become greenish from admixture of bile and in some instances shows a little blood. The bleeding is never sufficient to be of importance in itself. The vomiting continues for several days or even a week or more. The tongue is very heavily coated and may be swollen. Thirst is severe and the older children complain of the bad taste of the mouth. The abdomen is distended and there is tenderness to pressure over the epigastrium. The bowels may be constipated at first, but there is often a diarrhea later. The urine is scanty, high-colored, of high specific gravity, and contains urates or uric acid. The constitutional symptoms at the onset may be severe or slight. The temperature, if high at the beginning, soon falls and thereafter rarely exceeds 101° F. The pulse in the severer cases may be rapid and small. After the first few days the repeated vomiting, the restlessness, and severe thirst are the prominent symptoms. The affection runs its course in a week, as a rule, but unless care is taken the disease may be protracted much beyond this. Herpes labialis is not infrequent in older children.

The follicular inflammation of the stomach is, as already stated, very rare indeed and presents no peculiarity in its course beyond the fact that in the nature of things recovery will be much slower. It is no more likely to be attended with hemorrhage than the simpler form of inflammation.

The membranous gastritis is a pathological curiosity which most often gives no symptoms of its own and is recognized only at autopsy. It is conceivable that shreds of membrane might be vomited, but, so far as known, they have never been observed.

Of the symptoms of gangrenous gastritis nothing is known. In the single case alluded to there were no symptoms pointing to an unusual affection of the stomach.

**Diagnosis.**—This is usually determined by the course of the affection. The distinctive points from an acute indigestion have already been pointed out. As in that affection, one may fear the onset of one of the acute infectious diseases; especially are typhoid fever and meningitis to be remembered, but a few days' observation usually renders the nature of the affection clear. If the cause of the disturbance can be discovered, especially if this lie in the matter of feeding, the diagnosis can more readily be ventured.

**Prognosis.**—The prognosis is generally good. In weak infants, however, an attack of acute gastritis may be quickly fatal, or the infant may be left so exhausted that it gradually fails. The majority of the cases recover promptly under good care. When this is lacking the affection may become chronic. The prognosis in the toxic cases will depend upon the quantity of the poison taken and the promptness of treatment. Even when they recover the children are likely to be left with cicatricial stenosis of the esophagus or deformities of the stomach which will in the end prove fatal.

**Treatment.**—During the early stages this is to be conducted exactly on the lines laid down under acute gastric indigestion. If the temperature is high a sponge bath (water at 85° to 90° F.) for ten minutes will lower the fever and help to quiet the patient. If the vomiting persists, lavage of the stomach is the best of remedies. It may be repeated once or twice daily if necessary. For the relief of the thirst small bits of ice may be given to be held in the mouth or in the youngest patients a teaspoonful of cool water. In the severe cases small amounts of water, 60.0–120.0 c.c. (2–4 oz.), may be given by the rectum and repeated frequently, if the administration does not greatly excite the patient. Early attempts to feed the patient are more likely to do harm than good. Feeding is to be begun as indicated in the preceding chapter. The use of milk should be postponed for several days, and when it is resumed it should invariably be given as whey or much more diluted than the child had been previously having it. Barley-water or lime-water should be used as the diluent to prevent the formation of thick curds. Once milk has been satisfactorily begun it should be increased very gradually day by day until the patient is getting the normal amount. Freedom from vomiting, the return of the appetite, and the condition of the bowels should be our guides in making increases in the feeding. Any return of the symptoms is a signal for further dilution of the milk.

In cases of corrosive poisoning the use of the tube must usually be avoided, both on account of the spasm of the pharynx and esophagus and of the danger of furthering a perforation. If the patient has not already vomited, water should be given in large quantities, together with the proper antidote, which will probably have the effect of causing vomiting, and at the same time neutralize the poison. If the antidote is not at hand, there should be no delay in giving the water. Later, milk, oils, or albumen-water may be given freely. After the first few hours the treatment must be on the lines of any acute gastritis, except that washing the stomach is not advisable, and that morphine is required for relief of suffering. It should be given hypodermically. The combination of bismuth, cerium, and soda, given in the section relating to Gastric Indigestion, may be employed with advantage, or bismuth alone, 0.324–0.650 gm. (5 to 10 grains) every two hours. The severer cases should be treated with every care. The sick-room should be light and well aired. The patient should have at least one tepid or warm bath daily, depending upon his general condition; the bowels should be moved once daily; complete rest and quiet should be enjoined. Unless care is taken in all details one relapse may follow another until the acute condition has become chronic and ultimate recovery considerably delayed.

#### CHRONIC GASTRITIS.

Chronic Gastritis, Gastric Catarrh, or Chronic Vomiting is one of the most frequent of the disorders of digestion met with in infancy. It is a question whether in some cases there is an actual inflammation of the

stomach or whether the disturbance is not purely functional, but the distinction is not of practical importance. Chronic gastritis is, in most cases, associated with a similar disorder of the intestine and colon. As the recent researches of Pawlow have emphasized for us, the process of digestion cannot be properly separated into several independent acts; it is a continuous process, the proper performance of each step being essential to that which is next in order. Thus the best stimulus to antestinal digestion is the outflow of normal chyme from the stomach into the intestine. Imperfection in this lessens the normal stimulus to the intestine, and in turn impairs the intestinal digestion. So a chronic gastritis cannot long continue without disturbing the functions of the intestine, which may in turn give symptoms, but practically the stomach continues to be the source of most trouble and we can best consider the cases under this heading.

**Etiology.**—This disorder is seen in infants who are improperly fed and usually badly cared for. Infants on the breast do sometimes develop chronic gastritis, but very rarely. Among the poor the matter of the care of the children seems to be of almost as much importance as the food in determining their welfare. Lack of sunlight, bad air, poor food, irregularity in feeding, and exposure to cold and wet may all play a part in inducing such disorders as gastritis. The early giving of tea and coffee or liquors may be met with in some cases. The presence of some constitutional disorder which lowers the tone of all the tissues, such as rickets, syphilis, tuberculosis, or anemia, may be an indirect factor. Cardiac disease or chronic affection of the lungs or liver which will produce a chronic venous congestion of the stomach may induce a chronic catarrh. In convalescence from any of the acute infections, the functions of the stomach are impaired and a chronic gastritis may be easily developed. It is said that repeated attacks of acute gastritis may beget a chronic condition, but this is doubtful unless the cause of the acute attacks is still active in the intervals.

In older children chronic gastritis is usually the result of the persistent use of indigestible foods (pastry, pickles, pies, candies, etc.), or bad habits of eating (eating too rapidly, eating at irregular hours, etc.), or exercising violently immediately after eating, etc.

**Pathology.**—The gross changes to be observed in the stomach in these cases are hardly in proportion to the gravity of the disease. The stomach is nearly always somewhat enlarged, the mucous membrane shows few rigae, but appears smooth, may be injected in places, and is regularly covered with more or less tenacious mucus.

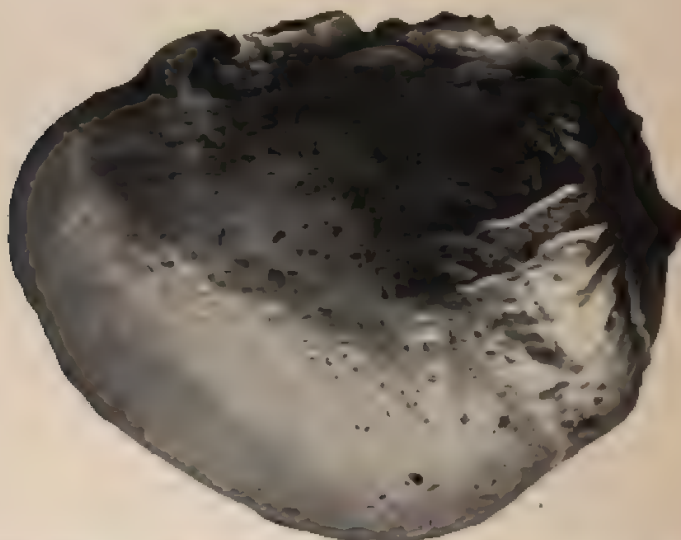
The advanced stages of chronic gastritis with much connective tissue in the wall of the stomach I have never seen in infants or children. The solitary follicles of the stomach may be enlarged. Microscopically, the stomach walls show more or less infiltration with round cells about the tubules, with secondary degenerative changes in the epithelium of the tubes (Fig. 39). Similar changes may be found in the intestine or colon.

**Symptomatology.**—Owing to these morbid changes in the stomach wall the secretion of hydrochloric acid and pepsin or pepsinogen is



definitely impaired; the process of digestion is therefore delayed; the food remains in the stomach much longer than it normally should—even six or eight hours—ferments and decomposes with the production of gas. The stomach, therefore, gets none of the normal rest between meals; is constantly dilated more and more by the accumulating food, mucus, and gas, so that the tendency is constantly toward a worse condition. The early and predominant symptom of chronic gastritis is vomiting. This at first may occur only rarely and be small in amount. It steadily increases in frequency and amount until it becomes more or less constant, the child vomiting after every feeding, upon the slightest movement or apparently without cause. With the onset of the vomiting the child ceases to gain normally, then begins to lose weight and loses steadily,

FIG. 39



Follicular ulceration of the stomach, a rare lesion accompanying chronic gastritis. Stomach of a child, eight months old, turned inside out and stuffed with horsehair. Cardiac end above, showing rugae

the subcutaneous fat disappearing from the body, the muscles becoming flabby, the skin dry, loose, and of a sallow tint; the face thin and pinched; the eyes sunken, but clear and bright; the fontanel depressed. In short, the child presents the picture of marasmus so familiar to all. The tongue is usually coated; the breath may be foul; the appetite is capricious, usually impaired, but it sometimes remains surprisingly good; the bowels are constipated, but may be loose if the functions of the intestines are also impaired. The abdomen is usually distended with gas, and is therefore markedly tympanitic, especially in the epigastrium. As the symptoms increase the child becomes very restless and fretful, crying more or less of the time, and getting very little quiet sleep. Gradually the strength fails until death ensues.

In advanced cases the children become greatly emaciated and lie as though lifeless; the feet and hands are cold, the pulse almost imperceptible, the respirations feeble and shallow; they cry only when disturbed; they take very little or no nourishment, and yet vomit from time to time the acid mucus. In this state they may linger for weeks before life ceases altogether, and from even this exhausted condition proper care may rescue them.

In older children the vomiting is not so pronounced, but is still the marked feature of these cases; the tongue is coated; the abdomen prominent and tympanitic; the bowels constipated. The children are very peevish and fretful; they may increase in stature, but the muscles are poorly developed and their vitality is very much impaired.

The vomitus in these cases at first consists only of food; later, as the vomiting increases in frequency, it comes to consist more and more of sour-smelling, acid mucus, with but little food. The analysis of the gastric contents or vomitus may or may not show the presence of hydrochloric acid. It will regularly show the presence of acetic, butyric, lactic, and other organic acids, resulting from the abnormal fermentation going on in the stomach.

**Diagnosis.** There is little difficulty in these cases. The history of the case, the character of the vomiting, and the results of physical examination are distinctive. A chronic meningitis might produce similar vomiting, but would be readily recognized by other signs. The important point in this relation is to search these cases of chronic vomiting for evidences of pyloric obstruction, for it has been clearly established that a certain number of them suffer from an hypertrophic obstruction of the pylorus, which is amenable to treatment by operative measures. (See Congenital Hypertrophy of the Pylorus.)

**Prognosis.**—Chronic gastric catarrh is always a serious affection in infancy. Undoubtedly many infants through this affection become marantic and ultimately die. Many more are so weakened by it that they fall ready victims to the diarrheal diseases of the summer or the bronchitis or bronchopneumonia of winter. Under proper care, however, the prospect of recovery is good and the infant may grow up into a vigorous adult. In later childhood the affection is much less serious as regards life, but almost always entails some failure of development, either in stature or in the tone of the muscular and nervous system. Good care and perseverance will, however, in many cases restore the child to perfect health.

**Treatment.**—The treatment of these cases must be both general and special. These children should have abundance of sunlight and air, and yet they should not be allowed to become chilled by cold. In summer they should live out-of-doors. During the cold months they should be out several hours daily, if this is compatible with keeping the circulation in a proper state. They should be dressed warmly and wear a flannel band and woollen stockings. The feet should be especially watched to make sure that they are always warm. Great care should be exercised to prevent infants being made wet by the vomitus; a towel should be kept



folded under the chin, if necessary, and changed as often as it becomes wet. Napkins when wet or soiled should be changed promptly. Each of these details will add to the comfort and welfare of the child. In the marantic cases a thorough rubbing and massaging once daily with olive oil or cocoa-butter aids nutrition and helps the circulation. The custom of using cod-liver oil for this purpose, because it is supposed to be absorbed and act as a food, has no sound basis in physiology or experience, and is extremely objectionable because of the odor.

The question of the regulation of the diet in these cases is a most difficult one, especially in the cases of breast-fed infants, and yet of the utmost importance. We occasionally see breast-fed infants who are vomiting persistently and failing in consequence, yet the analysis of the milk shows no error in its composition to satisfactorily explain the disturbance. The only thing possible in such case is to resort to a modification of cows' milk. If, on the other hand, definite irregularities can be found in the breast milk, and these can be corrected by changing the mother's mode of life or feeding, then we may hope for improvement in the infant's symptoms. Thus, if the mother's milk is deficient in fat and over-rich in proteids, we can usually, by feeding her more fat—*i. e.*, giving cream, butter, and meats freely, and enjoining exercise—correct this irregularity and so help the child; or we may resort to a manœuvre which I have found to serve the purpose in several instances, namely, have the mother remove one-half to one ounce of milk from the breast before allowing the infant to nurse. The first part of the milk, it is well known, contains less fat and more proteid; the latter part of the milk is richer in fat and poorer in proteid, which is just the part which is desired in such case. This plan also has the advantage of reducing the quantity that the infant can get at any one nursing.

If, however, the milk is over-rich in fat, the withdrawal of cream, milk, butter, and perhaps meats, from the mother's diet, will reduce the fat to nearer normal. But at the best we find that the amount of control which we can exercise over the breast milk is slight, and results in most cases are unsatisfactory. Even in the matter of control of nursing time, which one would assume would fairly well determine the amount of milk a child would get at a nursing, careful observation has shown me that the amount of milk taken in a five minutes' nursing may vary greatly at different hours of the day. In a nursing baby we should, therefore, analyze the milk or have it analyzed and endeavor to correct any irregularities discoverable. Nursing should not be permitted more frequently than once in three hours, at least one nursing being omitted during the night, and the nursing time should be reduced one-half at first. If these measures bring a definite improvement they may be persisted in, the nursing time being gradually lengthened again until the infant is getting all he desires. In most cases, however, we shall be driven to secure a wet-nurse or resort to artificial feeding.

In artificial feeding we have the great advantage of being able to control exactly the composition of the food, the hours of feeding, and the amount given at any one time.

In a case of chronic gastritis in an artificially fed infant, a change to the breast may be of the utmost value, and wherever it is feasible a wet-nurse should be tried. Sometimes trial must be made of several before a satisfactory one is obtained, the difficulty lying either in the composition of the woman's milk or in the digestive powers of the infant.

If artificial feeding must be resorted to or relied upon then the proportions of the food must be made very low and the quantity of the feeding reduced greatly in amount. A guaranteed milk, or a milk the freshness and cleanliness of which are assured, should be secured and, to begin with, diluted with 9 parts of a 5 per cent. milk-sugar solution (made by dissolving 1 ounce of milk-sugar in 20 ounces of water). This would give a mixture containing 0.4 per cent. fat, 5.5 per cent. sugar, and 0.4 per cent. proteids. An ounce of such food may be given once in three hours at first, and the quantity gradually increased, if it agrees with the infant, until nearly the normal amount is taken. Then the strength of the milk may be gradually increased by diluting a less number of times. The upward progress must be made slowly. Any attempt to rapidly increase the strength of the milk is sure to be followed by a return of the vomiting, and it is quite surprising on how small a quantity of a weak food an infant will get along well, if only the food is properly digested. If raw milk cannot be taken the milk may be peptonized and then diluted. If this is well borne the dilution is gradually lessened, and when the infant has taken this for several days the duration of the peptonizing process may gradually be shortened until the infant is able to take raw milk. It seems to be an advantage to use whatever digestive power the infant has, but I have known of instances in which the peptonization has been required throughout the first year.

In severe cases it may be that milk in any form cannot be borne and for a time we are forced to resort to other foods. Fresh beef-juice may then be used, one to four teaspoonfuls diluted with an equal part of water, chicken-broth in quantities of one to four tablespoonfuls, or the preparation known as peptonoids or panopepton, one or two teaspoonfuls diluted four or more times with water, for a feeding. It is to be remembered that the latter preparations contain about 20 per cent. of alcohol, and are therefore stimulants as well as foods. Valentine's beef-juice is also at times a serviceable preparation. After a day or two on such substitutes the peptonized milk should be tried again. Whey is also an excellent and valuable preparation for trial in these cases; or in children over six months of age barley-water or rice-water may be used. These latter preparations have the advantage that, if the infant will retain them, after a day or two milk may be added to the whey or barley-water, beginning with a single teaspoonful to each feeding, and increasing the quantity day by day according to the indications.

All of these foods are best given cold, and in bad cases it may be found wise to put aside the bottle and give all food for a time from a teaspoon. When an infant refuses food altogether its life may be saved by feeding it regularly through a stomach tube for a day or two. In all cases the greatest care should be taken of the infant's mouth, the

nurse being instructed to carefully cleanse the mouth with a 2 per cent. solution of boric acid after each feeding. Unless this is done a stomatitis or thrush is quite likely to develop and further complicate the case, if it does not prove fatal.

From time to time one sees cases of chronic gastritis, in which, after long trial, a proprietary food has been found which the infant takes and retains, but upon which it will not thrive. In such cases it is a good plan not to try radical changes of the food, but to add milk or cream gradually to it, exactly as we would add it to whey or barley-water. If the infant can be kept comfortable it is usually an easy matter to get it to gain by such means. The key to success is the very gradual increase in the strength of the food, after comfort has once been secured.

In cases of great exhaustion it is of the utmost importance to maintain the body heat and improve the circulation. Hot-water bottles should be kept constantly at the feet. Dry friction of the extremities is also advisable from time to time. If collapse occur a hot mustard bath should be given, the whole body being immersed until reaction is excited and the skin becomes flushed.

For the direct relief of the vomiting, stomach washing should be employed. It has the advantage of not only removing the mucus and decomposing food, but of also stimulating the normal secretion of the mucous membrane.

A solution of sodium bicarbonate, 4.0 gm.-500 c.c. (a drachm to the pint), should be used at a temperature of 100° F. The washing should be repeated daily at first, later every second or third day.

Medicinal treatment is of little service. The great majority of gastric sedatives have no effect whatever. Starr, however, recommends the use of Fowler's solution in the following form for a child of three months:

R—Liquor potassii arsenitis . . . . .	1.00 c.c.	(℥ xvj).
Sodii bicarbonatis . . . . .	1.56 gm.	(gr. xxiiv).
Aque menthæ piperitæ . . . . .	q. s. ad 100.00 c.c.	(℥ij).

Sig. 4 c.c. (one teaspoonful) in a little water, t. i. d.

Tincture of nux vomica may be given in the same dose, to improve the appetite and stimulate the muscular action of the stomach. A small graduate should be used to measure each dose. The household teaspoon varies so greatly in its content that much more than the intended dose may be given. By actual measurement nine teaspoonfuls have been found, in some cases, equal to 60 c.c. (2 ounces).

If constipation develops in the course of treatment it is best to move the bowels by the use of a gluten or glycerin suppository or a simple enema of soapsuds.

In older children the same general plan of treatment must be followed, and greater difficulties may be encountered by reason of the unwillingness of the children to submit themselves to the necessary régime. The general hygiene of the child must be attended to. As much time as possible should be spent in the open air. In those of school age it may be necessary to forbid attendance. Moderate exercise should be secured, with care that exercise is not carried to the point of overfatigue. The



sleeping-room should be well aired, and the child should be in bed by eight o'clock. The morning bath should be as stimulating as possible. With sensitive, weakly children the use of cold water is always objected to. The mother is therefore instructed to give the child a cleansing bath at whatever temperature is comfortable to it, determining the temperature by a thermometer; then, while the child stands in the tub, to sponge it rapidly with water a degree or two colder, and follow this with a vigorous rub. Day by day the temperature of the water used for sponging is gradually lowered, until the child is getting a bath that brings a vigorous reaction. With a little firmness this can always be accomplished.

Three meals a day are sufficient, the heaviest meal being given at noon. For the breakfast and supper, milk should be the mainstay, given at first diluted with plain water, barley-water, or Vichy water, at least one-half. Dry toast or zwieback may be given with it, or graham biscuit, not the flat crackers which are often used. Soft-boiled eggs or fish may be allowed later. The dinner should consist of clear soups without condiments, meats, and later the simpler vegetables, spinach, celery, cauliflower, peas, etc. All pastry and sweets should be forbidden.

Thorough mastication of the food required, and no other fluids than milk or water allowed. Dilute hydrochloric acid, 0.30-0.60 c.c. (5 to 10 drops), and tincture of *nux vomica*, 0.06-0.18 c.c. (1 to 3 drops), may be given after each meal with advantage, but here, as in the case of infants, reliance must be put upon diet and life rather than in medicines.

Stomach washing cannot be employed in these cases, but instead large draughts of warm water may be given an hour before meals. It seems to be an advantage to have the water sipped, rather than swallowed rapidly.

For constipation in these cases small doses of *casearia sagrada*, 0.065-0.130 gm. (1 to 2 grains) of the extract, or 2.0 c.c. (5ss) of the aromatic fluid extract, may be given at night, or the regular use of the familiar mist, *rhei et socke comp.*, 4-8 c.c. (5j ij) t. i. d., p. c.

A flannel binder should always be worn; the feet should be kept warm, and all exposure to wet and cold guarded against.

#### DILATATION OF THE STOMACH.

**Etiology.**—Dilatation of the stomach in infancy and childhood arises from causes similar to those operative in adult life. In general they are:

1. Obstruction to the passage of food from the stomach, usually occurring at the pylorus, and resulting in distention of the organ from the retention and decomposition of the food. The most frequent cause of such obstruction is an hypertrophy of the pylorus, which will be treated separately. Other causes are mentioned in literature, such as congenital strictures or obliteration of the duodenum or pylorus; strictures of the pylorus from the scars or ulcers; volvulus high in the small intestine, etc.; but they are all extremely rare and practically beyond our powers of diagnosis.

2. Weakening of the muscular wall of the stomach. This occurs from a number of constitutional causes, especially rickets, syphilis, tuberculosis, or severe anemia. It develops also to a greater or less extent in most cases of chronic gastritis. It is this form which presents itself practically and deserves consideration.

**Pathology.**—The apparent size of the stomach, as seen in autopsies on children, varies greatly, and depends to a considerable extent on whether the organ is contracted or relaxed at death. The actual size and capacity, of course, increase rapidly from birth onward. The normal capacity at birth averages from 30–45 c.c. (1 to 1½ ounces), at three months from 120–180 c.c. (4 to 6 ounces), at six months about 180 c.c. (6 ounces), and at twelve months from 240–300 c.c. (8 to 10 ounces). The capacity of a dilated stomach may greatly exceed these figures. Holt reports a child three months old with a stomach capacity of nine ounces; another four and one-half months old with a capacity of ten ounces; and an extreme case of a two-weeks-old baby with a stomach holding seventeen ounces. Apart from the dilatation these stomachs usually show the evidences of a chronic gastritis.

**Symptomatology.**—The symptoms are those of a chronic gastric catarrh. The infants present the usual picture of chronic vomiting, with resulting failure of nutrition. The vomiting is rarely of such large quantities as are seen in adult life. The vomitus may or may not contain hydrochloric acid, but does show lactic acid and other products of abnormal fermentation, and in some cases yeast and sarcinae. The condition is recognizable by the results of physical examination. The abdomen is distended, particularly in the epigastric region; this part of the abdomen being sometimes quite full, while the remainder is flat. In an emaciated child the outlines of the stomach may be seen through the abdominal wall. If the greater curvature is at the level of the umbilicus, or below, we may be quite sure that the stomach is dilated. Gastropexia without dilatation of the stomach is practically unknown in childhood. Percussion over the empty stomach gives a loud, resonant tympanitic note, which may enable us to outline the organ without artificial distention. It is frequently possible, by passing a stomach tube, filling the stomach with water, and then emptying it, to demonstrate an abnormal capacity. To accomplish this the water must be allowed to flow in very gently; rapid introduction will excite vomiting much before the stomach is really full. Comparison of the percussion notes of the full and empty stomach will also enable one to locate its borders. If these methods are not satisfactory the stomach may be washed out and then gently distended with air by attaching the bulb of a Davidson's syringe to the tube. Succussion may be obtained by shaking the body of the child, or clapotage by placing the tips of the fingers of both hands upon the epigastrium, and giving alternate quick taps or thrusts; but these signs are not of value alone, they may be obtained over a normal organ.

**Diagnosis.**—As already said, this rests upon the physical signs. The only probable source of confusion is a dilated colon, but care in the observations suggested above will enable one to differentiate the two conditions.



**Prognosis.**—Dilatation of the stomach is not in itself a grave condition, although it is difficult to correct. It may, however, prove a very serious matter in an infant attacked by severe disorder of the lungs or heart. The pressure of a large, distended stomach may then serve to greatly embarrass respiration or the action of the heart, and may determine a fatal issue in a case where the prognosis would otherwise be good.

**Treatment.**—The treatment is practically that of a chronic gastritis, as given above. Special care should be taken to limit the quantity of nourishment as much as is consistent with proper nutrition, to avoid the use of any articles of food which might increase the abnormal fermentation, and to relieve the stomach of the accumulations of food, mucus, and gases. For this latter purpose washing the stomach regularly once a day is, when possible, of great value. As a tonic to the muscular coat tincture of nux vomica is recommended in small doses, as given for chronic gastritis. The ordinary gastric sedatives are of little value.

If there is an underlying constitutional condition, especially rickets, that should receive attention and treatment, so far as is possible under the circumstances.

If there is reason to suspect an organic stricture of the pyloric region, an exploratory operation might be done, and in any case where palliative treatment had failed, if the child's condition permitted it, a gastro-enterostomy should be considered as offering a possibility of recovery by providing better drainage.

#### CONGENITAL HYPERTROPHY OF THE PYLORUS.

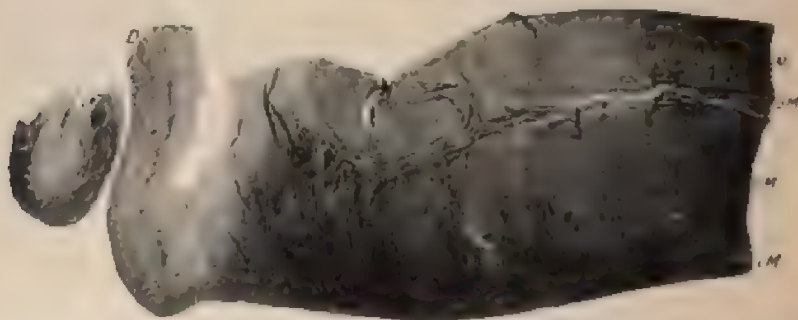
Since the original observations of Landerer (1879) and Maier (1885) called attention to the occurrence of a congenital obstruction of the pylorus, many observations have been made which serve to show that the condition must occur more frequently than has been supposed. Among the most valuable recent contributions to the subject are those of Thomson, of Edinburgh, and Still, of London, to whom I owe my knowledge of the affection.

**Etiology.**—This is entirely a subject of theory at this time. Thomson inclines to the view that the hypertrophy is produced by a derangement, probably from faulty development, of the nervous mechanism which regulates the contraction and relaxation of the pylorus under appropriate stimuli. As a result of such derangement the muscle of the pyloric sphincter is overworked and consequently hypertrophies. Others hold that the condition is simply an error in development. Ashby reports a case in which hypertrophy of the pylorus was found in an infant which had been operated upon for imperforate rectum. It is to be remembered also that instances of complete closure of the pylorus by failure of development have been recorded.

**Pathology.**—In the normal infant's stomach the pylorus is represented only by a slight thickening in the wall of the tube, indefinitely marked

off from the adjacent parts. The wall of the pylorus, in a normal infant of five months, was found by Still to measure 1.7 mm.; in two, of ten months, 2.6 mm. and 2.5 mm., respectively. The lumen of the pylorus, under one year, Still found to admit a probe 3.5 to 4 mm. in diameter. In cases of congenital hypertrophy the pylorus forms a cylindrical tube from 2 to 2.5 mm. in length, with muscular walls of about 5 mm. in depth, and a calibre varying from the normal to an aperture that will admit only the finest probe (Fig. 40). Sometimes after the removal of the stomach the contents can hardly be pressed through the stenosed pylorus. The lumen of the sphincter after death will vary with the degree of spasm present at the time of death. The thickened pylorus may form a tumor which can be felt during life. The remainder of the stomach is dilated to a greater or less extent and may be coated with thick, tenacious mucus. The esophagus is dilated in some cases.

FIG. 40



Hypertrophic pyloric stenosis, section through the pylorus and adjacent duodenum: *M*, mucous membrane; *SM*, submucous coat; *CM*, circular muscular coat; *LM*, longitudinal muscular coat, *D*, intestine. (Dr. John Larkin's preparation from Dr. Downing's case.)

Microscopically the thickening of the pylorus is found to be due to a great increase in the muscle of the sphincter. The connective tissue may also show some increase, but not so much as the muscle. The mucous membrane may show some swelling and engorgement, but is usually not greatly changed.

**Symptomatology.**—The first symptom of this condition is vomiting. This does not begin immediately after birth, and is usually slight at first, but gradually becomes more frequent and more severe. It may not occur until the infant is a month old, but once it begins it is usually persistent. Such cases, however, do show a remission of symptoms for a time, the improvement being probably due to a relaxation in the element of spasm, which undoubtedly figures in a degree, at least, in these cases. The vomiting at first occurs some time after the administration of food, but gradually the stomach becomes more irritable, until toward the end any food taken is promptly rejected. The vomitus consists of the food, more or less modified, and mucus; bile is never present. Usually a variety of foods is tried in succession, but without

result so far as the course of the affection is concerned. The vomiting is sometimes very forcible. It seems to depend more upon the quantity of food than upon its composition. At first only large quantities of food bring it on; later, smaller and smaller quantities excite it. The ordinary gastric sedatives have no effect upon the disturbance, but in some cases washing out the stomach and giving the food by gavage may temporarily arrest it. The infants suffer from all the symptoms of chronic vomiting: scanty urine, constipation, emaciation, etc. The question of the operations of the bowels calls for attention. One may be told that the infant is not constipated, as it has a movement daily, but observation will show that these movements are very small and contain little or no food. Toward the end of life the movements may consist almost wholly of mucus. The abdomen is distended in its upper part, particularly in the epigastric region; the remainder of the abdomen is retracted and permits satisfactory palpation.

There are two other physical signs of great importance: (1) The presence of peristaltic movements in the dilated stomach. It may require repeated careful observations to detect these peristaltic waves. Whether they can be excited by irritation of the epigastrium, as they can be in adults, is not known. (2) The presence of a small, movable tumor in the region of the pylorus. The mass formed by the hypertrophied pylorus has been both seen and felt in some cases. This sign should be carefully sought, as it is practically conclusive of the nature of the trouble. The course of the affection is usually steadily progressive, although, as noted above, remissions do occur in some cases. The duration of life in the fatal cases has been three weeks to six months.

**Diagnosis.**—The essential points in the diagnosis are these: (1) The infant is born healthy, and, without apparent cause, begins to vomit at within the first few weeks of life. (2) The vomiting persists despite treatment or change in the nourishment and the greater portion of the food is rejected. (3) There is constipation and the stools are formed mostly of bile and mucus. (4) There is progressive emaciation. (5) Peristaltic waves are visible in the dilated stomach. (6) A small movable tumor is visible or palpable in the region of the pylorus. The last two points are apparently conclusive of the diagnosis. In the absence of both one would feel great hesitation in venturing an opinion, especially in an artificially fed child, for many of these, we know, vomit persistently on one food and yet improve promptly when the food is changed. In a breast-fed child the other symptoms would be of much more import.

**Prognosis.**—There is little doubt that this condition is fatal in nearly all cases, unless the stenosis is relieved by operation. Finklestein reports three cases from Prof. Heubner's private practice which recovered under palliative treatment. The diagnosis in these cases is a little uncertain. Batten has, however, reported a case in which, although the infant was greatly emaciated and a tumor was observed, recovery followed without operation. The cases regularly end fatally within a few weeks. Osler has resurrected from the earliest medical publication

of this country an account of a case in which the patient, a boy, lived to the age of five years.

**Treatment.**—So long as the diagnosis is in doubt the condition should be treated on the lines of a chronic gastritis. Washing out the stomach and feeding the patient by the tube seem to be the measures of greatest value. If operation is inadvisable or is not permitted this treatment may be continued, although our present experience gives little reason to hope for success. If the diagnosis is established operation should be urged. The question of the exact operation to be done lies between: (1) Gastroenterostomy, which is the operation recommended by most surgeons. It has been done in 10 cases with 5 cures. (2) Pyloroplasty, which has been done in 4 cases, all successful. (3) Loreta's operation, which has been tried in 12 cases with 7 recoveries. The number of recorded cases is still too small to justify conclusions as to which operation offers the best prospect. The question will probably be decided by the preference of the surgeon undertaking it. The most complete discussion of this subject is the article by Ashby in the *Traité des Maladies de l'Enfance* of Grancher and Comby, 1904, second edition.



## CHAPTER XI.

### ACUTE GASTROENTERIC INFECTIONS.

THE terminology employed in relation to the acute diarrheal diseases of children has always been unsatisfactory. For many years all these affections were thrown into one great group under the designation of summer diarrheas. This term was given up because the affections in question were not by any means limited to summer, and also because under one name were included diseases of evidently different etiology, lesions, and symptoms. The attempt was then made to classify these several affections on the basis of their anatomical lesions. Only greater confusion resulted, for it was soon found that this led to meaningless subdivision, many varied lesions being found to be associated with the same clinical symptoms, so that in order to reach a satisfactory classification an autopsy was required in every case. Inasmuch as the use of infected or impure milk is the most frequent apparent cause of these disorders, and especially as the phenomena of one group at least of the cases are identical with those produced by certain potent poisons, known to be developed in milk by bacterial action, Vaughan, of Ann Arbor, and others have proposed to classify all these acute gastroenteric infections under the title of Milk Infections. So far as the particular group of cases described as cholera infantum is concerned, there is ample support for this view, but the extension of the conception to cover all the cases in question seems unwarranted. In the light of our present knowledge it seems probable that there are other means of contagion quite as important as milk. No one scheme has been found to meet the needs of the situation. The hope was entertained that when the etiological factor in these several diseases was discovered we would be able to classify them more simply and satisfactorily on that basis. The investigations of the last two years have apparently established the Shiga bacillus in this relation to a large number of the acute diarrheas of childhood, but for the present these investigations have added still further to the prevailing confusion. Instead of finding that this bacillus is the cause of a certain definite group of lesions in the gastroenteric tract or is associated with disease of a definite clinical type, we find that it occurs with lesions varying from the mildest catarrhal inflammation to a croupous inflammation and severe ulceration, and also that the clinical types of disease in which the bacillus occurs are equally varied. The situation is still further confused by reason of the fact that as the Shiga bacillus was originally identified in relation to adult dysenteries, investigators have taken to applying the term dysentery to the diarrheal diseases of child-

hood in which the bacillus is found, although many of these diarrheas have not the clinical symptoms, the presence of blood and mucus in the stools, with which we have all been accustomed to associate the term. Furthermore, the term dysentery has, for some years, not been in use in writings dealing with diseases of children, or, when used, it has been limited to the colitis excited by the presence of amebæ. Altogether, therefore, the situation, as it confronts us at the present time is a very complicated one, and I have thought best to follow the classification of Holt, which is familiar to most of us, is consistent with itself, and is the most satisfactory at present known to me, rather than to attempt a new classification which could only be tentative.

The general causes of the diarrheal diseases of infancy and childhood have already been discussed. In the present chapter we are to deal with those acute diarrheal affections which are especially common in summer and which are now admitted to be due to bacterial infection; not that these affections are by any means limited to the summer, for they do occur from time to time during the winter, but every summer in these latitudes is marked by a wave of these diseases, which might be justly called an epidemic. The wave begins when the daily atmospheric temperature reaches or surpasses an average of 60° F., such temperature being necessary to the general growth and diffusion of the bacteria concerned, and continues until the falling temperature of late September or the first of October puts an end to this condition. Ordinarily, the wave begins in June, early or late, according to the temperature conditions, rises rapidly to a maximum in July, continues high, but with variations, during July and August, and gradually subsides during the latter part of August or in September. The amount of rainfall or the humidity seems to exercise no definite influence upon the course of the outbreak. It must not be understood that the activity of bacteria is the only factor in the production of these diseases. The bacteria are admitted in most cases, at least, with food, most especially with milk. The ways in which milk becomes infected are therefore of the greatest importance in the spread of these diseases. Practically, the only pathogenic organism known to occur with any frequency in milk as it leaves the cow's udder is the tubercle bacillus. Certain varieties of streptococci are regularly found in all milks, but their pathological importance is disputed. The harmful infections and resulting changes in the milk occur after it leaves the cow's udder, either in the process of milking, in transportation, keeping, or preparation for feeding, or in the feeding process itself. The measures for avoiding infection in milking and the keeping of milk have, of recent years, been worked out most carefully, and have proven effective in keeping down the bacterial content of milk. The essential point is absolute cleanliness at every stage of the process, secured by the careful cleansing of the cows' udders, the milker's hands, the sterilization of all vessels used, etc. The milk should immediately be chilled to a temperature below 50° F. and kept below that point. In the preparation of the milk and the feeding of infants the principles of asepsis should

be followed and even the best of milk should be pasteurized or sterilized during the summer months. The effectiveness of these measures in reducing the mortality from the diarrheal diseases is admitted by all. As in any infection, whatever lowers the vitality of an individual predisposes to an attack, so in this relation bad hygiene, constitutional disease, and especially any previous disorder of the alimentary tract are to be regarded as factors of importance. These infections may occur in any of the earlier years of life, but are much more common in infancy.

In the case of the most acute and severe of these disturbances, cholera infantum, it seems highly probable that the disease is produced, as Vaughan maintains, not by the presence of bacteria alone in the infected milk, but also by that of larger or smaller quantities of the soluble poisons, toxins or leuconaines, that are produced in culture media by the growth of bacteria. It may be that these poisons are already present in the milk, when it is given, or they may be produced by the continued action of bacteria after the milk has been consumed. Tyrotoxicon, a poison of the class first found in cheese, has been isolated from a sample of milk which had produced a severe choleraform diarrhea in a child. This tyrotoxicon, says Vaughan, will in animals produce the symptoms and lesions of cholera infantum. Various other toxins have been isolated from cultures of bacteria which have been found in the intestine in cases of acute diarrheal disease and have been proven pathogenic in animals. In Vaughan's opinion these toxins are probably as numerous as the bacteria that produce them. The suddenness of the onset of cholera infantum in many cases in children previously well, the resemblance of the symptoms to those produced by any acute irritant poison, such as arsenic, the relatively rapid subsidence of the symptoms and recovery of the patient certainly support the idea that it is these toxins rather than the bacteria themselves which are the immediate and direct agents in this disease. The conception certainly seems to explain the difference both in symptoms and in lesions observed between cholera infantum and the more subacute or chronic cases comprehended under the term ileocolitis.

**Bacteriology.**—Until the recent discovery of the presence of the *Staphylococcus* the results of a great deal of laborious work which has been done upon the bacteriology of the acute diarrheal disorders of infants and children have been very unsatisfactory. The rectum of the newborn is sterile, but after a few hours bacteria begin to appear and increase rapidly in number. These are chiefly bacteria of putrefaction. Escherich found fairly regularly a saprophytic bacillus, a non-pathogenic chain coccus, and the bacillus subtilis. In the stools of nurslings after the beginning of milk feeding the bacillus lactis aerogenes and the bacillus coli communis are constantly found in addition to many other putrefactive bacteria. In all diarrheal conditions the numbers and varieties of bacteria are greatly increased. Booker worked out a considerable number of these, but without being able to demonstrate a specific relation between any of the bacteria and the conditions in which they were found. Booker, Baginsky, and



Escherich have laid especial emphasis on the abundance of streptococci in certain cases, and German writers recognize a distinct condition of streptococcic enteritis, but the establishment of a distinct type of disease due to streptococci has not been recognized by American clinicians. In 1902 Duval and Bassett, pupils of Flexner, working in the Thomas Wilson Sanitarium near Baltimore, were able to demonstrate in the stools of a considerable proportion of children suffering from acute diarrheal diseases the presence of the Shiga bacillus. This bacillus was first isolated and demonstrated to be the cause of epidemic dysentery in man by Shiga, a Japanese investigator. Later, it was found by Flexner and Strong and Musgrave in the dysenteries of soldiers in the Philippine Islands. It was next found by Martini and Lenz in Germany, and by a number of investigators in our own country in isolated cases or localized (institutional) outbreaks of dysentery. Since 1902 the findings of Duval and Bassett has been confirmed by further studies on the part of Duval, Wollstein, Howland and La Fétra and many others. Its presence is not limited to any one previously recognized clinical type of disease. It has been found in simple fermentative diarrhea and in the more severe types of disease associated with more or less severe lesions of the colon and lower part of the small intestine, ileocolitis. Its presence in the intestine has also been demonstrated to be associated with a specific agglutinative reaction in the blood of the patient, similar to the Widal reaction obtained in typhoid fever. While the Shiga bacillus is found in a considerable variety of different clinical conditions, it has been demonstrated with greatest regularity in the diarrheas attended with fever and the presence of mucus and blood in the stools; in other words, the cases most closely resembling the dysenteries of adults. The proportion of cases of this kind in which its presence can be shown has varied remarkably in different investigations. The variations seem to depend, to some extent, upon the skill and experience of the bacteriologist making the investigation. Park maintains that in every case of acute diarrhea of the dysenteric type (that is, with blood and mucus in the stools) this bacillus should be found. It is recognized that there are at least two distinct cultural varieties of the Shiga bacillus: one, known as the true Shiga bacillus or the alkaline type, does not ferment mannite; the other does ferment mannite and is known as the acid or "Harris" or "Flexner-Manila" type. The latter is the type chiefly found in infantile diarrhea in New York. Quite a number of instances of infection with both organisms have been reported.

The agglutination reactions of the several varieties of the organisms have proved most confusing, and as the belief in the causal relation of the bacillus to the diarrheas in which it is found rests upon the demonstration of a specific reaction between the bacillus and the blood of the patient, conservative bacteriologists are not yet thoroughly satisfied that such a relation exists—that is, they do not believe that the Shiga bacillus has been proven to be the exciting cause of these infantile diarrheas or dysenteries. The fact that the Shiga bacillus has been



found in some few instances in normal stools has some weight in the argument, but we are to remember that the diphtheria bacillus is also found in normal throats, and yet no one longer questions its relation to diphtheria. However, in view of all these facts I consider it best to hold to the accepted classifications, limiting myself to a statement of the case of the Shiga bacillus as investigations have thus far revealed it.

#### **SIMPLE GASTROENTERIC INFECTION OR SUMMER DIARRHEA.**

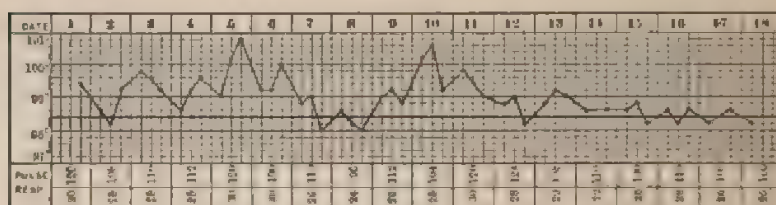
**Etiology.**—As the general causes concerned in the production of diarrheal diseases in infants and children have already been discussed, the essential points may be summarized here. 1. Age: From birth through the second year these affections are common, and are much less frequent in later years. 2. Bad hygienic surroundings, particularly residence in the crowded tenement districts of our great cities. 3. Artificial feeding. 4. Irregular and improper feeding, especially over-feeding, and the use of bacteria-laden milk. 5. Hot weather. 6. Bacterial infection. The specific organism is not known. Some regard streptococci or the bacillus proteus vulgaris as the offending bacteria. Others think that bacteria normally resident in the intestine, such as the bacterium lactis aerogenes and the bacillus coli communis, may, under certain favorable conditions, assume a pathogenic activity. Undoubtedly, a certain portion of these cases are included in the category of those produced by the Shiga bacillus.

**Pathology.**—Few of the cases are fatal except in those already suffering from chronic affections of the stomach and intestines, the lesions of which have been confused with those of the acute process. This is essentially an acute catarrhal inflammation of the stomach and intestinal tract. The stomach is usually distended with gas and food. Its walls are coated with mucus and possibly show irregular patches of congestion. The upper part of the small intestine is usually normal. The lower ileum shows some congestion and a little swelling of the mucous membrane. Peyer's patches may be swollen and hyperemic. In the colon similar changes are found. The ascending and transverse colon may be distended with gas. The congestion is mainly upon the rugæ and is more marked in the lower part of the colon than above. The solitary follicles may be swollen and their margins marked out by a zone of congestion. The mesenteric lymph nodes are swollen and may be slightly hyperemic. The contents of the intestine are thin, watery, green or yellowish in color, show undigested food, and are foul-smelling; there is usually but little mucus in the feces.

**Symptomatology.**—The symptoms may appear gradually or suddenly. The gradual onset is most often seen in those who are already suffering from chronic disorder of the alimentary tract, and are weakened or marantic therefrom. In these cases the movements of the bowels become more frequent, are at first yellow, later green, or brown in color, and foul

in odor. The patients have a little fever; they are peevish, fretful, and restless, especially at night. The abdomen is distended with gas and the infants suffer from pains, causing them to cry sharply at times and lie with the legs drawn up. Vomiting may occur in these cases but is not marked. Such children lose weight rather rapidly, the skin and tissues generally become pallid and relaxed, and more or less prostration results. After two or three days under proper care the symptoms subside and the infant returns to its former condition, or if neglected the symptoms of an ileocolitis gradually develop and the disease runs the protracted course characteristic of that condition. The sudden onset is more often seen in children previously well. The first symptom is usually vomiting, and on taking the temperature it is found to be elevated,  $102^{\circ}$  to  $103^{\circ}$  F., even  $105^{\circ}$  or  $106^{\circ}$  F. The vomiting is repeated. At first the vomitus consists simply of the food present in the stomach, then of mucus and water, and later bile may appear. In from four to six hours after the onset the diarrhea begins. At first the stools are yellow and contain undigested food, then they become green, with whitish

FIG. 41



Temperature chart of a case of enterocolitis in a child aged sixteen months. Shiga bacillus, acid type, isolated from stools: recovery.

lumps or curds; they are often mixed with gas, and consequently frothy. The tongue is coated with a white fur, the thirst is usually severe, and the infants take eagerly whatever is offered, only to vomit promptly thereafter. Restlessness and fretfulness are marked, particularly at night. In the severer cases the temperature may reach  $103^{\circ}$  to  $105^{\circ}$  F., the vomiting and purging be quite persistent, but they have not the serous character seen in cholera infantum, and the extreme prostration and severe nervous symptoms of this affection are wanting. The stools may be very frequent, even twenty being passed in a day, and often with pain. More or less erythema and excoriation appear about the anus and buttocks from the irritation of the discharges. The stools are usually large, are expelled with considerable gas, are gray, green, or brown in color, and foul. Weight is lost rapidly, the prostration may be marked, the fontanel becoming depressed and the eyes sunken, the pulse rapid and feeble, the muscular relaxation marked. The temperature is usually quite irregular, varying from  $99^{\circ}$  to  $103^{\circ}$  F., or even higher. Often after a large evacuation the temperature will fall several degrees. The abdomen continues distended and full from the presence of gas in the

intestines. After two or three days of severe symptoms improvement may be shown by less fever, less frequent vomiting, and less prostration. The diarrhea usually continues for some days. Progress toward recovery may be rapid, but may be interrupted by any new mistake in feeding or by the occurrence of very hot weather. In most cases the symptoms gradually subside and the infants make good recovery. In some instances after the beginning of improvement, the fever persists, the diarrhea continues, mucus and perhaps blood appear in the stools, and the cases run the course of an ileocolitis.

In the worst cases or in infants already weakened by preceding disease no improvement occurs; the fever, vomiting, and diarrhea continue; the infant passes into a condition of stupor and dies from exhaustion or convulsions.

**Diagnosis.**—It is well known that almost any acute disease in a child may begin with acute gastroenteric symptoms, especially scarlet fever, pneumonia, and tonsillitis. The diagnosis of ileocolitis must be reached by the absence of any of the characteristic signs of these diseases.

It is more difficult, as a rule, to tell in just which category of the acute disturbances of the alimentary tract to classify a given case. From acute indigestion the cases are distinguished by their occurrence in summer, higher fever, greater prostration, severer vomiting and diarrhea, and the abundant, foul-smelling stools. Many of the cases still more closely resemble those classified as ileocolitis, and, as has been said, many go on to develop the lesions and symptoms of that condition. As a rule, the cases of acute gastroenteric infection are shorter in duration, improvement coming within three or four days, but the chief difference lies in the absence from the stools of the blood and mucus characteristic of the ileocolitis. Some days of careful observation may be required to determine the diagnosis.

**Prognosis.**—Even in severe cases the prognosis is usually good. The prospect of recovery is most influenced by the age of the child, the severity of the onset, and the promptness with which appropriate treatment is instituted. An acute gastroenteric infection occurring in an infant under three months of age or in an older child already weakened by previous disease is often fatal. Many of them succumb at the very onset, still more die in the course of this affection itself or from a resulting ileocolitis. Prompt and effective treatment often plays an important part in deciding the outcome.

**Prophylaxis.**—This infection belongs especially to the summer season, but is favored by any disturbance in the digestive tract. The hygiene and diet of infants during the summer should be regulated with especial care. They should be kept out-of-doors practically all day long, but they must not be exposed to the direct heat of the sun. It is best by all means, when possible, to send infants and children from the city to the country for the summer months. There is no reason to prefer the sea-side to the mountains, except that the changes of temperature from day to night are usually less sudden and severe at the sea-shore than at the higher altitudes. Either is vastly better than the stifling heat of



an overcrowded city. Care should always be taken on cool nights to see that infants are properly covered. If circumstances do not permit a prolonged stay away from the city, the daily excursions that are conducted by so many charitable organizations in large cities may be of service.

Infants and children should be bathed frequently during the summer, at least once a day, and better twice, morning and evening. They should always be given water in abundance, cooled but not iced. Even the youngest infants will take water with advantage on hot days. If possible to keep an infant on breast-feeding during the summer it is always best to do so. Weaning should, unless absolutely necessary, be avoided until the fall. If artificial feeding is resorted to, milk of assured purity should be used, and care should be taken that it is as fresh as possible. City milk is often two days old. Whatever the milk, pasteurization or sterilization should be employed during the summer months; in the latitude of New York this is best done from May 1st until October 1st. Of next importance is care not to over-feed. Children, especially infants, should never be urged to feed during hot weather. Lack of desire for food is, as a rule, good evidence that food should not be given. Greater care than usual should be taken in making any increases in the strength of the food. On very hot days it is best to reduce the amount of each feeding one-third or more and supply the deficiency by the addition of water. Every disturbance of digestion should be regarded seriously and effort made to correct it, lest it open the way to serious infection.

**Treatment. Hygienic.**—Fresh air is of the utmost importance in the management of these cases. The patients should be kept in the open air, but protected from the sun, all day long, and doubtless many would do better if their nights also were spent out-of-doors. But care must be taken during the hours of the night to see that the infants are sufficiently covered to keep the feet warm. If it is possible, it is most advantageous to send these cases promptly to the country, either mountains or sea-side, so long as they get fresh air. Twice a day the infants should be sponged with cold water and the skin kept carefully powdered around the buttocks and genitals to prevent the excoriation which is so common. Diapers should be changed promptly when soiled and should be disinfected, either by antiseptic solutions or by boiling, before being used again. There is little evidence of the transmission of the infection from one child to another, but in view of the abundance of bacteria in the stools and the possibility that the individual himself may be reinfected, disinfection of the diapers is advisable. In hospitals it should be insisted upon. Quiet and rest should be secured as far as possible. Where many of these children are gathered in large hospital wards the crying and fretting of one or more will keep all awake and interfere with the sleep that is of great importance to recovery.

**Dietetic.**—In nurslings. The infant should be taken from the breast and kept from it until the acute symptoms of the onset have subsided. If the vomiting is marked it is best not to attempt feeding at all for



twenty-four hours. Boiled water should be given cold in small (teaspoonful) quantities, until tolerance shows that more can be retained. If feeding seems to be necessary albumen-water or whey may be given, one to two tablespoonfuls every two hours, not oftener, until the temperature is lower and the vomiting and diarrhea somewhat lessened. Then the breast may be allowed, the quantity taken being restricted by limiting the nursing time to three or four minutes at first and permitting nursing only once in four hours, with whey or albumen-water in the interim. Water may be given at any time to relieve thirst, unless its administration provokes vomiting. If return to the breast aggravates the symptoms, nursing should be entirely stopped and will probably have to be given up entirely. A second trial may be made after another interval of twenty-four to forty-eight hours' feeding with the whey or albumen-water, and if this results badly there should be no hesitation in changing to artificial feeding entirely or securing a healthy wet-nurse. If breast feeding has to be abandoned the case will have to be treated exactly as though artificial feeding had been originally employed; that is, in attempting to feed we should begin with very dilute foods, then use cows' milk highly diluted, and so on.

In the artificially fed we begin by cutting off all food, especially milk, for twenty-four or forty-eight hours. The preparations known as liquid peptonoids or panopepton, diluted three or four times, and given cold in 4.0 c.c. (teaspoonful) doses every hour or two, will often be retained better than anything else. While of doubtful food value, their considerable percentage of alcohol (about 20 per cent.) makes them valuable as stimulants. We may test the retentive power of the stomach with these and then try weak foods of greater value—albumen-water, whey, chicken-broth, beef-juice, malted or cereal milk, and dextrinized barley-gruel. Whatever food is given must be tried in small quantities, about one-half what the infant would ordinarily receive, and in the case of such foods as malted milk, in a strength suited to the digestion of an infant half the age of the patient, or even less. Albumen-water or whey is usually borne fairly well, even by the youngest infants. To an infant of three months we may begin with 15 c.c. to 30 c.c. (one-half to one ounce) every two hours, and increase the quantity to two or three ounces gradually. Fresh beef-juice may be given in quantities of 15 c.c. to 30 c.c. (one-half to one ounce) as an alternative feeding; special care is needed that this is prepared from untainted meat. The greatest difficulty may be experienced in some cases in getting the infant to take any food whatever, and we may have to try one food and then another before we find one that the infant will take and digest. The condition of the stools as well as the course of other symptoms must be watched for guidance as to the digestion and assimilation of the foods given. Beef-juice and albumen-water may give offensive stools. After one or more of these substitutes have been used for several days, if the temperature has subsided and the stools have shown definite improvement, both in number and in their consistency, milk may be tried. If whey has been found to agree it is

usually best to begin the administration of milk by adding a single teaspoonful of milk to each feeding of whey. If milk alone is to be used it must be given at first diluted with many times its volume of a 5 per cent. solution of sugar of milk. Thus, for an infant under three months of age we may use a dilution with nine parts of such sugar solution, which would give us a milk mixture containing approximately 0.4 fat, 5.4 per cent. sugar, and 0.4 proteid. If this is well borne the dilution may be diminished gradually to give us constantly increasing percentages of fat and proteid. This may be accomplished by substituting a 6 per cent. or 8 per cent. cream for the plain milk and diluting as before. If plain milk is found to be not digested the milk may be peptonized. Holt recommends peptonization for as much as two hours to ensure the complete digestion of the proteid. Wherever possible it is preferable to use diluted milk or cream, in order that we may know exactly the composition of the food given and regulate our increases accordingly. There is no doubt that in most instances in infants the dilution of milk with a cereal water, barley-water preferably, renders it more digestible, especially if the cereal be dextrinized by the addition of one of the diastatic ferments. If we can once get the infants to digest even small quantities of milk it is usually possible by very gradual increases to get them to gain in weight. Our first aim should be to secure the comfort of the infant, by giving a food that can be digested. Too great haste in making increases in the strength of the food, in order to secure an increase in weight, will only result in increasing the disturbance and delaying recovery. It may be necessary to be content with little or no gain until the return of cooler weather improves the atmospheric conditions and revives the patient.

In any case the progress is usually slow and marked by more or less frequent relapses, sometimes due to changes in the food, again to increase in the atmospheric temperature, or other unfavorable conditions. Whenever any food is given a trial, several days are usually required before we can tell definitely whether or not it is being digested; changes should not be made too rapidly. Many a case that looks hopeless may be saved if a good wet-nurse can be secured. If breast milk can be digested, progress will be more rapid and satisfactory than with any other form of feeding. Unfortunately it is difficult to secure the desired nurse under any conditions, and especially so when the infant is desperately sick.

*Medicinal.*—In the beginning of treatment it is best to give a dose of calomel or castor oil. If the stomach is not disturbed castor oil is preferable, 4.0 c.c. (1 drachm) for a child under one year of age, 8.0 c.c. (2 drachms) for one over a year, and 15 c.c. ( $\frac{1}{2}$  ounce) for children of three or four years. In cases where vomiting has been repeated, castor oil will usually be rejected; we then give 0.065 gm. (a grain) of calomel in divided doses; 0.01 to 0.015 gm. (gr.  $\frac{1}{4}$  to gr.  $\frac{1}{2}$ ) every hour till 0.065 gm. (1 grain) is taken, to a child of one year; 0.12 gm. (2 grains) is given in the same way to older children. Later in the course of the disease, whenever there is an increase in the symptoms, especially

if the stools become more frequent and show more undigested food, it is best to repeat the dose of oil or calomel. In any case when a change of food seems desirable it is best to clear the intestine in this way. For the control of the diarrhea itself an almost endless list of intestinal antiseptics has been brought forward and each has found more or less advocacy; but two or three have proven sufficiently satisfactory to continue to enjoy general usage. Bismuth undoubtedly holds the first place. The subnitrate, subcarbonate, salicylate, and subgallate have all been recommended. The subnitrate and subcarbonate are given in large doses, 0.650 gm. (10 grains) or more every two hours after the feedings. The subgallate or salicylate in doses of 0.130 to 0.260 gm. (two to four grains) every two hours, after feedings. These may be given in powders, but for administration to infants or young children it is better to suspend them in some such prescriptions as the following:

R—Bismuth. subnitratiss . . . . .	8.0 gm.	(5j).
Acacia . . . . .	2.0 "	(gr. xxx).
Tragacanth . . . . .	2.0 "	(gr. xxx).
Aque . . . . .	ad 120.0 c c	(3iv).—M.

Sig.—4-8 c.c. (one or two teaspoonfuls) every two hours.

Or,

R—Bismuth. salicylatis . . . . .	4.0 gm.	(5j).
Acacia . . . . .	2.0 "	(gr. xxx).
Tragacanth . . . . .	2.0 "	(gr. xxx).
Aque . . . . .	ad 120.0 c.c.	(3iv).—M.

Sig.—4-8 c.c. (one or two teaspoonfuls) every two hours.

Of these undoubtedly the subnitrate is still preferred and seems to be as useful as any. Large doses are required to be of any service. Salol also is often employed in doses of 0.12 to 0.24 gm. (2 to 4 grains) every four hours.

R Salol . . . . .	4.0 gm.	(5j).
Ol. olive . . . . .	10.0 c.c.	(3iv).
Acacia . . . . .	2.0 gm.	(gr. xxx).
Tragacanth . . . . .	2.0 "	(gr. xxx).
Aque . . . . .	ad 120.0 c c	(3iv).—M.

Sig.—4-8 c.c. (one to two teaspoonfuls) every four hours.

(The olive oil is necessary to dissolve the salol.)

Salicylate of soda may be used in solution.

R—Sodii hypsulphitis . . . . .	0.75 gm.	(gr. x).
Sodii salicylatis . . . . .	4 to 8.00 "	(5j to ij)
Aque menthae pip. . . . .	ad 120.00 c c.	(3is).—M.

Sig.—4-8 c.c. (one to two teaspoonfuls) every four hours.

The hyposulphite of sodium is added only to prevent the mixture changing color and becoming black.

The more complex antiseptics, such as  $\beta$ -naphthol,  $\beta$ -naphthol bismuth, tannin, etc., have not found any general acceptance. The simpler our prescriptions can be kept in these conditions the less irritating the medicines will be, and, as a rule, the better will they be borne. Any medicine which causes vomiting should be promptly stopped lest it do more harm than good. One of the difficult problems in these conditions is that of the use of opium. For a long time opium in some form was added



to nearly every mixture used. Lately, this has been entirely given up and opium, if administered, is given alone, the better to regulate the dose and administration. Undoubtedly, the diarrhea can be checked by the use of opium in any form, but not always with benefit to the patient. Opium should be used only for one of two purposes: (1) to relieve pain, or (2) to check excessive peristalsis due to the intestinal irritation or inflammation. It is to be remembered that the diarrhea is to some extent a protective process, ridding the system of products of fermentation which, if retained, do harm. It is, therefore, easy, by entirely stopping the action of the bowel by opium, to do harm to the patient. Opium is best given either in the form of the camphorated tincture (paregoric) in doses from five to twenty drops, repeated every one, two, or three hours, until the desired effect is produced. Small doses may contribute much to the comfort of a patient and help to recovery. Dover's powder may be used as a substitute, in doses of 0.01 to 0.015 gm. ( $\frac{1}{4}$  to  $\frac{1}{2}$  grain), repeated in a similar way. In severe cases Holt recommends morphine hypodermically, 0.0006 gm. (gr.  $\frac{1}{1000}$ ) for a child a year old. For great restlessness or in conditions where convulsions seem to be threatening, no other remedy can be so effective.

Stimulants will be required in many cases to meet the prostration and exhaustion of the disease. Alcohol in the form of whiskey or brandy is usually best. Either may be given to the amount of 15 to 30 c.c. ( $\frac{1}{2}$  to 1 ounce) daily to a child one year old. Each dose must be given diluted from four to six times with water. It is best to give small quantities, say 10 to 30 drops, every hour or two. Much larger amounts can be given if necessary. In cases of severe vomiting ice-cold champagne may be retained when any other form of alcohol is vomited. It may be given in teaspoonful doses, diluted two or three times with water. For extreme prostration whiskey may be given hypodermically, 10 to 15 drops diluted with sterile water, or we can resort to hypodermoclysis, as described on page 240.

Lavage of the stomach and colon may both be of great service. In the early stages, washing out the stomach will serve the purpose of emptying it of some of the toxins; it will also check the vomiting, and it may be resorted to at any time when vomiting is frequent. Plain water, normal salt solution, or 4 gm. (1 drachm) of sodium bicarbonate to 500 c.c. (1 pint) of water are to be used for this purpose. In most instances it is advisable to leave 15 to 30 c.c. (1 or 2 ounces) of fluid in the stomach to appease the thirst. It will often be retained under these conditions when rejected in any other way. Lavage of the colon serves to remove decomposing and irritating material from the bowel. It should always be employed at the outset and may be repeated three or four times in twenty-four hours, later once or twice daily will be sufficient. The temperature of the water used should be about 85° to 90° F.

It is to be remembered that collapse can be increased by rectal irrigation, and care should be taken in weakly children to raise the temperature of the water and shorten the duration of the process. Each washing is to be continued until the colon is thoroughly emptied and



the water returns clear. The body temperature will be lowered in proportion to the temperature of the irrigation, and these irrigations may be regularly employed as one means of controlling high temperatures in these conditions. The irrigation should, as a rule, be stopped when the temperature returns to normal, otherwise the washing may prove sufficiently irritating to the colon to continue the diarrhea some time longer than would otherwise be the case.

When lavage of the stomach cannot be employed large draughts of water may be given, the resulting vomiting being depended on to clear the stomach. If the vomiting has already been repeated or excessive, washing or the giving of much water may be dispensed with.

The active treatment of these cases may be summarized thus:

1. Stop all feeding for twenty-four to forty-eight hours, allowing water freely.
2. Clear the stomach and intestinal tract by washing stomach and colon and by giving calomel or castor oil.
3. When feeding is resumed adapt it to the digestive power of the patient.
4. For the control of the diarrhea rely mainly on the feeding. Subsidiary measures are: (a) washing out the colon daily; (b) the use of intestinal antiseptics or antifermentatives.
5. Make all increases in food cautiously, watching especially the general condition of the patient and the condition of the stools as guides.
6. Attention to the details of hygiene and fresh air and quiet are most valuable aids to our other measures.

#### CHOLERA INFANTUM.

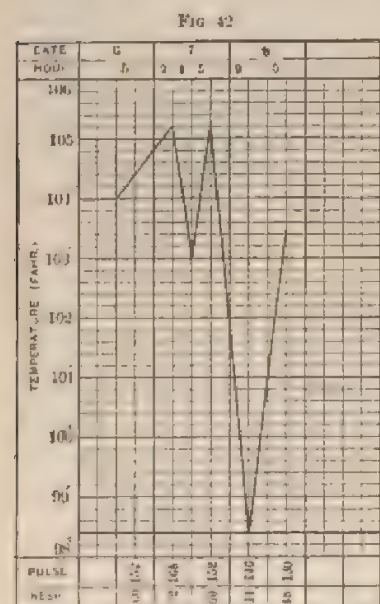
**Etiology.**—The general considerations in this regard have already been stated. True cholera infantum is a disease of children under the age of three years. It is practically unknown in breast-fed children. It occurs regularly at the height of summer and is not seen during the winter months. The view that the disease is an acute poisoning with the toxins produced in milk by bacterial growth seems to me to best meet the facts of the case. These toxins are probably present in the milk at the time of ingestion, but may also be elaborated within the body. The specific organism or organisms are not yet known.

**Pathology.**—The symptoms of the disease are out of all proportion to the lesions found present in the body after death. The bodies of the dead are notably reduced in proportion to the duration of the disease; the abdomen is retracted; the tissues are pale and relatively bloodless; the eyes are sunken. The lesions in the stomach and intestines are surprisingly slight. Usually the whole alimentary tract is pale and bloodless, having a washed-out appearance as it is usually stated. The contents of the intestinal tract in the upper part are thin, yellowish or grayish watery fluid containing particles of food and little mucus; in the colon the contents may be the same or may be greenish in color, and contain more flocculi and mucus. Their odor is described as musty, not foul. The mucous membrane of the intestine may be cloudy and show a slight loss of epithelium on the surface; the solitary follicles may

appear a little swollen. Microscopically, in addition to this superficial loss, there is some small round-cell infiltration of the mucosa and submucosa. The intestinal contents under the microscope show particles of food, a little blood, and epithelium in single cells or in masses. The lesions in other parts are not important. The lungs are pale anteriorly, posteriorly congested, and with small areas of collapse. The kidneys are large and pale, with slight cloudy degeneration of the epithelium of the tubules. The serous membranes are dry and sticky. The blood is rather thick and dark.

**Symptomatology.** The onset of the disease is very sudden and its development rapid. An infant previously suffering from some mild digestive disorder, or it may be in apparently perfect health, suddenly begins to vomit and shows a rise of temperature. The vomiting is soon followed by diarrhea. The vomitus is at first the usual contents of the

stomach, later thin, watery fluid mixed with little mucus. The stools likewise consist at first of ordinary intestinal contents, but rapidly become thinner, green or gray or almost colorless, very watery, and with a musty odor. In the severe cases the vomiting and purging become almost incessant, the stomach will retain nothing, the bowels move fifteen or twenty times in twenty-four hours, and the infant shows a profound constitutional depression. Substance and weight are lost rapidly, as the tissues are drained by the serous discharges from stomach and bowels. The surface is cold, especially the extremities, while the rectal temperature mounts more or less steadily, in the fatal cases reaching 107° or 108° F. before death. The eyes are sunken, the pulse rapid and feeble, and the respiration shallow and weak. The children are limp from



Temperature chart of a fatal case of cholera infantum in a child eight months old.

exhaustion and muscular relaxation. Thirst is severe and distressing. The quantity of urine is greatly decreased. The abdomen is usually retracted and soft. The mental condition is profoundly affected. At first the infants are restless and fretful, but soon pass into a condition of partial stupor, in which they lie with sunken, up-turned eyes, tossing the head from side to side, trying to moisten their dry lips with parched tongues, and either entirely silent or moaning piteously. Wild delirium may at times occur. Convulsions are not uncommon.

By remissions in the vomiting and purging may occur, but in the majority of instances the downward progress is usually steady.

Most of the fatal cases terminate within forty-eight or seventy-two hours (Fig. 42). Some writers speak of an algid state with a subnormal temperature, but this I have never seen. In other instances the onset and course of the disease are not so severe. The fever develops, the vomiting and purging have the typical character, but are not so continuous, the constitutional depression is not so profound, the infants rally, and, in the course of a few days, the temperature falls, the emesis and diarrhea gradually lessen, and the infants convalesce normally. In other instances the infants rally partially, but gradually develop the symptoms and apparently also the lesions of an acute ileocolitis, which will be described later.

**Diagnosis.**—This is usually easy. The frequency and character of the vomiting and purging, taken with the fever and sudden collapse occurring in an artificially fed child under three years of age, are sufficient to stamp the picture clearly. The only other affection producing such symptoms is the true Asiatic cholera, from whose ravages we are happily free. In conditions which warrant a doubt a bacteriological examination of the stools for the specific organism would be required to settle the question. In some quarters the affection has been confused with sunstroke, but the much more rapid development of coma in the latter without the characteristic vomiting and purging easily distinguish the two. Some of the less severe cases closely resemble the acute ileocolitis, but are distinguishable by the difference in the vomitus and stools, by higher temperature, and more rapid recovery when once the severe storm of onset is passed. Some, indeed, of the cases go on to develop the features of an ileocolitis, as already observed.

**Prognosis.**—This is by all means the most serious and fatal of the acute diarrheal diseases of infancy. The great majority of the cases are fatal and are apparently little influenced by treatment. The possibility of recovery seems to rest rather upon the vitality of the patient and the severity of the poisoning than upon the treatment employed. As Holt observes, there is little ground for the assurance that the fatal result might have been averted had the physician been called sooner.

**Prophylaxis.**—The essential points in this regard have already been given above. The vital point lies in the condition of the milk given for food. Pure milk, properly kept, will never produce cholera infantum. Milk loaded with bacteria, and kept at temperatures permitting bacterial growth, may. It is also well to remember that apparently trivial digestive disorders may open the way for these acute disturbances or, at least, make the patient more vulnerable.

**Treatment.**—There are three chief indications: 1. To empty and cleanse the stomach and intestine. 2. To control the temperature. 3. To combat the collapse.

Medication by either of the ordinary routes and feeding are for the time out of the question. Food should be at once stopped. 1. The stomach and bowels are both to be cleansed by washing. Simple salt solution or solution of sodium bicarbonate, 4 gm. to 500 c.c. (1 teaspoonful to the pint) may be used for this purpose. The washing of



the stomach should be done with water at a temperature of about 100° F. The bowels may be washed with water at a temperature of 90° F. If the vomiting and purging continue, these washings may be repeated in four to six hours, but it is usually not advisable to continue them so frequently for more than one day. 2. The temperature is to be controlled by baths or packs. The bath is to be preferred because friction can be employed at the same time to keep up the circulation and prevent collapse. The infant is to be put in water at a temperature of 95° to 100° F. and then the temperature is to be gradually lowered to 85° F., such a bath to be continued for fifteen to thirty minutes. Constant friction,

FIG. 43



Method of hypodermoclysis. The bottle contains normal salt solution. The tube is first filled by "stripping" to start siphon action. Two needles are used, one being inserted on either side of the abdomen.

especially of the extremities, should be employed during the bath. Where the baths fail to control the temperature, rectal irrigation with ice-water has been recommended, the water being allowed to run in and out freely; but I have seen collapse produced or aggravated so often by such measures as to consider them more likely to be harmful than good. The baths may be repeated every two or three hours as necessary. In the intervals between the baths cold packs to the trunk may be employed, the infant being wrapped in a sheet wrung out of water at a temperature of 90° F. and water of the same temperature sprinkled over it



from time to time. In cases of marked depression it is well to leave the feet and legs out of the water and apply heat to the feet. Cold applications are to be employed on the head. 3. To meet the collapse stimulants are necessary, but cannot be given by the mouth or rectum; they must be introduced by the skin. Normal salt solution, 3 gm. to 300 c.c. (45 grains of salt to a pint of sterile water), is to be given by hypodermoclysis (Fig. 43). The ordinary siphon apparatus suffices for this purpose. The apparatus must be sterile. 200 to 300 c.c. can in this way be given at one time. Hypodermoclysis has the great advantage that it not only acts as a stimulant, but supplies the fluid which is so greatly needed in the tissues and to promote the excretions of poisons by the urine. The injection may be repeated at discretion in from four to six hours. Fluid is taken up from the loose cellular tissue with great rapidity under these circumstances. Holt especially recommends the injection of morphine, 0.0006 gm. (gr.  $\frac{1}{160}$ ), and atropine, 0.000075 gm. (gr.  $\frac{1}{800}$ ), as stimulants to the heart, and especially for the relief of severe nervous symptoms. Cardiac stimulants, whiskey, camphor, ether, may be given hypodermically also. Whiskey in 0.30 to 0.60 c.c. (5 to 10 minims) doses is to be given well diluted with hot water; ether may be used pure in like quantities. Camphor is to be dissolved in a sterile sweet-almond oil, 1 part to 10, and from 0.30 to 0.60 c.c. (5 to 10 minims) given at a time.

If with these various remedies we check the onward progress of the disease the vomiting and purging lessen and the nervous symptoms improve. So soon as the stomach permits we may begin the administration of iced champagne or brandy well diluted, 5 to 10 drops in a teaspoonful by mouth. As the tolerance of the stomach increases, ice-water may be given by mouth in increasing quantities. The more fluid that can be gotten into the system the better, but the return of vomiting will frequently check these measures. After twenty-four hours' improvement we may begin feeding with liquid peptonoids or panopepton, 2 to 4 c.c. ( $\frac{1}{2}$  drachm to 1 drachm) in water given every two or three hours. If this is well borne we may after twelve or twenty-four hours give whey, beginning with 15 c.c. to 30 c.c. ( $\frac{1}{2}$  ounce or 1 ounce), once in two hours, and gradually increasing the quantity. From this transition may be made to milk diluted as in acute gastric disturbances, and then we may gradually work back to ordinary feeding. If the diarrhea persists with the presence of mucus or mucus and blood in the stools the cases must be treated as ileocolitis.

## CHAPTER XII.

### THE DIARRHEAS OF INFANCY AND CHILDHOOD—DISEASES OF THE INTESTINES.

#### THE DIARRHEAS OF INFANCY AND CHILDHOOD.

THE most important of the illnesses of infancy and childhood are the disorders of the intestinal tract associated with diarrhea. The great part of the mortality of infancy is due to these diarrheal diseases, and many children who are not killed by them are left permanently impaired in stature and vigor and may suffer from digestive disorders for the remainder of life. In considering the cause of such disorders many factors must be admitted.

1. **Physiological.**—Relatively the alimentary tract of an infant is called upon for vastly more work than that of the adult. A healthy infant at the age of a year will take and digest from one quart to a quart and a half of cows' milk. An adult we find can be sustained by from two to four quarts. The weights of the two are to one another as 1 to 7 or 1 to 8. The infant for his weight is doing three or four times the digestive work of the adult. This greater activity of the digestive apparatus entails a greater sensibility to disturbing influences, so that the slightest change in diet or régime may in infancy be reflected in some intestinal disorder.

2. **Mode of Feeding.**—Breast-fed infants suffer much less from diarrheal diseases than those artificially fed. As a rule not more than 2 per cent. or 3 per cent. of the children suffering from summer diarrheas are breast-fed. It is not merely the differences in chemical composition of cows' milk and human milk that come here into play, for the cleanliness of the milk, its freshness, the care with which it is prepared and given, all serve to influence the result. It is generally recognized that since the poorer classes of the people have learned the advantages of sterilization of milk, the frequency of diarrheal diseases has steadily diminished.

In older children the eating of green fruit and unrestrained indulgence in candies, ice-cream, and soda-water serve to bring on these affections.

3. **Age.**—The period from the sixth to the eighteenth month is the period of life most afflicted by diarrheal diseases. This corresponds with the time of substitution of artificial feeding for the breast, or among the poorer classes the substitution of table feeding for the bottle. It is no uncommon thing among the poor to find children not yet a year old getting practically the food and drink of their parents, even to beer and berries in the summer season.

4. **Season.**—The diarrheal diseases prevail to a greater or less extent all the year round, and the summer is the time of special danger. The wave of mortality from infantile diarrheas begins in June, early or late, depending upon the atmospheric conditions, rises to a maximum in July or August, and continues with little change until the cooler days of September bring relief. The mortality also varies from year to year directly with the severity of the summer heat. The two summers of 1902 and 1903 in New York City were notably mild, and the ravages of the diarrheal diseases were correspondingly less.

5. **Surroundings.**—The tenement districts of our great cities suffer most severely from these affections, but they are not unknown in the country. The children of the well-to-do escape, for the most part, because they have the advantages of pure air, sunlight, etc., combined with due care in the selection, preparation, and giving of their food. The children of the poor suffer not only because they have not pure air and are surrounded oftentimes by filth, but still more by reason of their being fed on impure milk, which is prepared without care, and given in the way that involves the least trouble. The establishment of depots for the distribution of sterilized milk in our large cities has done much to lower the mortality among infants from these causes. In New York City the Strauss laboratories and the work of St. John's Guild have been of great value not only for their direct help in this way, but for their educational influence upon the poorer classes of the population. The emphasis that is being laid upon the necessity of watching the milk supply of our cities, not only that the milk be up to the standard in composition, but, much more, that it be clean, free from serious bacterial contamination, and that it be supplied to the consumer with as little delay as possible after milking, is exerting an influence all over the country, until even now in the smaller cities or towns one will find dairymen awake to the new demand for clean milk and endeavoring to supply it. When our tenement population can obtain clean milk for their children they will suffer less from diarrheal diseases.

6. **Care of Children.**—This is certainly a factor of great importance. It has not been clearly shown that the summer diarrheas are transmissible, but there is little doubt that they are. The demonstration of the presence of a specific agent such as the Shiga bacillus in a large proportion of these diarrheas certainly implies that the affection may be directly transmitted from one to the other. The prompt change of soiled diapers is to be enjoined, lest the child contaminate its hands and so infect itself or convey infection to others. Thorough cleanliness of the child's person should be enforced, and nurses or others handling these children should be cautioned as to the cleansing of their hands. Especially should anyone feeding an infant be careful of the cleanliness of the hands, so as not to contaminate the bottle or nipple, and thus infect a child. These precautions are particularly necessary in all large institutions or hospitals where numbers of children suffering from these diarrheal diseases are gathered together.

We cannot teach infants not to put their fingers in their mouths, and



unless their hands are kept clean, we cannot prevent their taking in bacteria that may do harm. It has been shown that tubercle bacilli can be carried under the nails in the dirt gathered from the floors of dwellings or the streets.

Most of all, the pernicious custom of giving infants "comforts" and such like objects to suck should be warred upon. When we see mothers picking these objects from the floor, the carriage, or even the street, and with a hasty brush of the hand restoring them to their children's mouths, we wonder how so many of the children survive the experience.

**7. Constitutional Condition.**—Children that are weakly from any cause, but especially those suffering from rickets, syphilis, or tuberculosis, or malnutrition in any form, are subject to these diarrheal diseases. For this reason children in hospitals or asylums are especially prone to these affections, and great numbers of them are carried off yearly by them. Even the slightest disorder of the gastroenteric tract in an infant or child is of importance, for the reason that during the summer it is very likely to become so much worse as to seriously affect, if not destroy, the individual's life. It is this fact which renders important the careful treatment of even the slightest gastroenteric disorder in the early years, a fact that also serves to explain the greater seriousness of these disorders among the poor, who regularly wait until the disorder has assumed a serious character before seeking advice or instituting proper treatment.

**8. Bacteria.**—Although investigation has shown that there were myriads of bacteria present in the intestine in these diarrheal disorders, it was not until recently that any specific relation could be established between any of these bacteria and the diseases in question. It now seems established that a bacillus of the colon-typhoid group, known as the Shiga bacillus, from the original discoverer, can be regarded as the specific agent in a considerable number and variety of these diarrheas. Just how large a part of our summer epidemics will be accounted for in this way it is too early to say, but observations made during the past two summers indicate that this bacillus is to be found in practically all the summer diarrheas in which mucus and blood are found in the stools, and in a certain proportion of the cases in which these constituents are absent. Of the life-history of this organism outside the body very little is yet known. The natural assumption is that infection takes place by means of water, milk, etc., but of this there has, as yet, been no scientific demonstration. How far the hopes that these discoveries will in the end reveal the modes of infection, determine the means of prevention, and possibly materially reduce the mortality from the diarrheal affections of summer are to be realized is altogether uncertain at present. (See page 227.)

The relation of the bacteria in milk to these disorders is an interesting question. It is well known that 1 c.c. of milk, as it comes to the consumer, contains from 5000 to 5,000,000 or even 10,000,000 bacteria. Of just what varieties this enormous total is made up but little is known, except that nearly all are non-pathogenic to man. The tubercle bacillus is the only pathogenic organism found at all frequently in milk. Diph-



theria and typhoid bacilli have been found in a very few instances. Streptococci are practically always present, but it is not known that the varieties of streptococcus found have any harmful effect upon the human organism. Although it has not yet been shown in just what way these multitudes of bacteria in milk affect the individual consuming it, it has been clearly shown that a high bacterial content is associated with conditions in the milk that render it harmful, that produce gastroenteric disorders, and is therefore sufficient ground for the rejection of such milk as food, particularly in the cases of invalids or children.

### SIMPLE DIARRHEAS.

By this term we designate the diarrheas which are marked by the frequent movements of the bowels, the stools consisting only of undigested food or food and water, without blood or mucus, and unaccompanied by fever or severe constitutional disturbance. A number of varieties are distinguished.

1. **Mechanical.**—Undigested food of any kind, such as fruits, nuts, green corn, and the like, may produce diarrhea, simply by acting as mechanical irritants, stimulating peristalsis and driving the intestinal contents through before digestion is completed. The movements in these cases are frequent and watery, and contain more or less undigested food, often plainly showing its original structure. A dose of castor oil or calomel, with some restriction of diet for a time, promptly cures these cases.

2. **Nervous or Reflex Diarrhea.**—That nervous excitement or emotion can produce a diarrhea is a fact known to all, and applies to children as well as to adults. The influence produced by dentition upon the intestines has been somewhat debated. There seems to be no question that the eruption of a tooth can produce a diarrhea which will last until the tooth is through the gum and then subside. Admitting this, one need not agree to the common belief that all the diarrheas of the period of dentition or of infancy are due to teething and should be permitted to run their course, because stopping the diarrhea would injure the child. A sudden chill or wetting of the feet may also excite a simple diarrhea which belongs in this class. The management of these reflex diarrheas consists simply in the treatment of the cause of irritation—as soon as that is removed the diarrhea ceases.

3. **Colliquative Diarrhea.**—Colliquative diarrhea is seen in certain of the infectious diseases, or in uremia. The diarrhea in these cases seems simply to be one of nature's methods of getting rid of poisons, just as we ourselves are accustomed to move the bowels freely in the effort to free the system from toxins. To a certain extent, therefore, these disorders are protective and not harmful, but they often run on to an extent that saps the patient's strength and greatly reduces him. It is then necessary to stop them. A severe diarrhea in the course of a pneumonia, for example, is always a grave symptom and seems often

to determine a fatal outcome. Whether this is simply another evidence that in such cases the system is overcharged with toxins and the individual is too poisoned to recover, or the diarrhea itself exhausts the patient, it is difficult to say. To check such a colliquative diarrhea we would change the nourishment to a simpler and more digestible form, such as peptonized and sterile milk, or withdraw milk entirely and use only beef-juice, barley-water, or whey, and give sufficient doses of opium to check the motions. The opium is best given in the form of Dover's powder, 0.00125 to 0.06 gm. (gr.  $\frac{1}{4}$  to gr. 1), which may be given every two or three hours to a child under two years, until the desired effect is produced. Purgative may be used instead, the dose for an infant a year old being from 0.60 to 1 c.c. (10 to 15 minims).

4. **Diarrhea from Drugs.**—Diarrhea may, of course, be excited by the use of drugs. There is certainly some ground for the belief that in nurslings diarrhea may be excited by the presence in breast milk of purgative drugs which the mother has taken.

#### ACUTE INTESTINAL INDIGESTION.

This affection is analogous to the acute gastric indigestion already described, both in etiology, lesion, course, and treatment. It may or may not be accompanied by gastric symptoms, but in this case the intestinal disturbance is the chief factor and dominates the clinical picture.

**Etiology.**—As in the gastric affection, the cause is nearly always some irregularity in feeding, such as the taking of too much food, or of indigestible food, food of bad quality, sudden changes in the dietary, etc. In breast-fed infants we find acute intestinal indigestion occurring in the infant in connection with disturbances in the mother's health, sometimes in relation to the menstrual period, in other cases attending unusual excitement or exertion on the mother's part. It may be that analysis of the breast milk in these cases will show some distinct changes in its composition, but often this is not the case. Not infrequently as the breast milk begins to fail, it becomes poor in fat, over-rich in proteids, and then excites an acute intestinal indigestion in the infant.

In artificially fed children this disturbance may be brought about by faulty composition of the milk, attempts to feed too large proportions of proteids, sometimes apparently also by too low proteids, by feeding milk that is laden with bacteria, or that has become changed by them. The early feeding of solid food, especially if that solid food be of an indigestible character, is a common cause in infants who are being weaned. The farinaceous foods by reason of the readiness with which they undergo decomposition are particularly likely to set up such a disturbance.

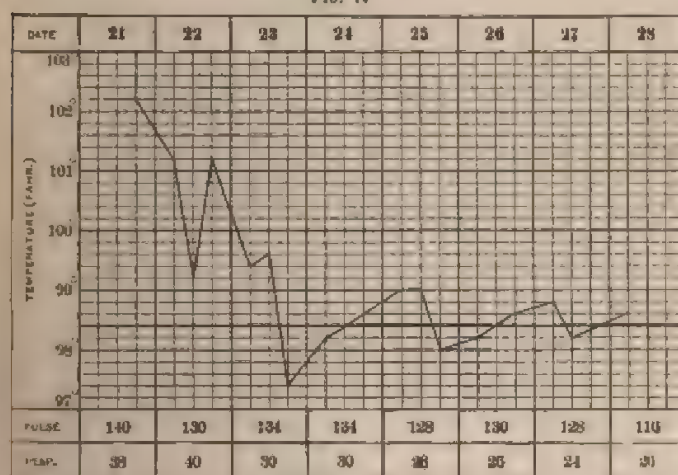
Exposure to wet or cold with resulting chilling of the surface and congestion of the internal organs is a factor of some importance. As is well known also the heat of summer so affects the digestive apparatus that during the summer an infant may no longer be able to digest a food

which he had previously been taking care of perfectly well, and the first evidence of this may be an attack of acute intestinal indigestion. Children of either sex and of any age are susceptible, but the greater proportion of cases occurs in those from six to eighteen months of age. It has already been pointed out that children whose vitality has been lowered by reason of any constitutional disease—rickets, syphilis, tuberculosis, or malnutrition of any form—are more susceptible than others to these digestive disorders.

**Pathology.**—We infer that in these cases there is a functional disturbance of the intestine without definite anatomical lesion, but the borderline between this and catarrhal inflammation is purely theoretical. Many writers in fact include acute catarrhal inflammation of the intestines under this heading.

**Symptomatology.**—The attack of acute intestinal indigestion is either sudden in onset or the symptoms develop gradually. In acute cases the

FIG. 44



Temperature chart of a case of intestinal indigestion in a child eight months old.

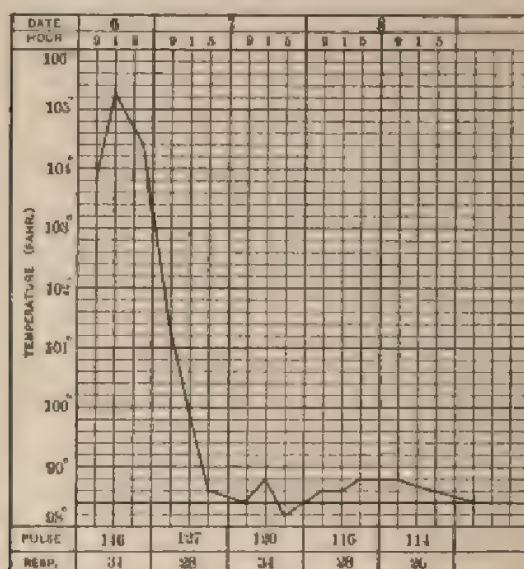
onset is marked by a sudden rise of temperature, 102° to 104° F. (Fig. 44), abdominal pain, restlessness, peevishness, disturbed sleep, rapid pulse, and more or less languor. In feeble children even convulsions may occur. The diarrhea may not appear until some hours after the onset of the attack. When it does occur the movements contain the undigested food (not infrequently the cause of the disturbance can be detected in the stools) and are very watery. In these severe cases there is usually some vomiting at the outset, but this quickly subsides. After the first day or two the temperature falls and the pulse becomes slower, but the diarrhea persists (Fig. 45). Usually the appetite is lost and the tongue coated white, but on account of the loss of water in the stools thirst is marked. The abdomen may be distended and tympanitic, but frequently remains flat. The urine is scanty and high colored. The child usually shows



marked prostration, the eyes are sunken, the face is pallid, weight is lost very rapidly, particularly in children previously fat, and there is a general muscular relaxation.

In the less acute cases the onset may be without temperature, marked only by restlessness, fretfulness, and crying from the abdominal pain; the diarrhea is not so severe and vomiting is less likely to occur. After the first day or two the course of the affection is much the same. The loose movements are most likely to follow feeding, each bottle or nursing being followed by one or two loose dejections. The character of the movements is characteristic. At first they are yellow with more or less undigested food. If the food is milk, the casein appears in flakes

FIG. 45



Temperature chart of a case of intestinal indigestion in a child aged ten months.

in the watery fluid, or in large, rough, white masses mingled with the yellow of normal feces. Soon the color changes to a green, in which the white masses of casein are conspicuous, or the whole stool becomes of a bright grass-green hue. The change in color, it has been shown, has been caused by the substitution of biliverdin for the bilirubin of normal feces, but the exact explanation of this substitution is not known.

The duration of an attack of acute intestinal indigestion varies from one or two days to a week. Usually the diarrhea gradually subsides and the other disturbances with it. Repeated attacks may lead to more serious intestinal disturbance, such as the ileocolitis to be described later, or a chronic intestinal indigestion.

**Diagnosis.** The course of the affection is characteristic. The character of the diarrhea taken with the other symptoms and the rapid



subsidence of the disturbance distinguish it from the more serious disorders of the intestinal tract. Until the diarrhea appears there is nothing to warrant a diagnosis.

**Prognosis.**—The attack is rarely fatal except in an infant already greatly enfeebled, but it is remarkable how rapidly an apparently healthy child, especially the large, fat, rosy baby, will fail under such an attack. It loses weight and is prostrated to an extent that it may require weeks to repair. This applies particularly to babies who are fed artificially on patent foods. If proper care is not taken one attack may open the way for a severe intestinal infection, or the persistence of the cause may lead to a chronic disorder.

**Prophylaxis.** In this is involved the proper regulation of the diet and life of a child, especially during infancy, and although a thorough consideration of the subject cannot be given here, there are two points which can properly be made: 1. That during the summer months the feeding of a child should be kept relatively low both in quantity and in proportions. Especially is this necessary under the conditions which prevail in cities like New York, where many of the children are sent to the country for the summer, and are thus deprived of the careful supervision which they enjoy the rest of the year. It is my custom to stop increases in the feeding about the first of June, and let the children pass the summer on the food which they have shown ability to digest up to that time. Even this may not be sufficient and further dilution may be required. If increases are made either in quantity or quality, they should be carefully watched. 2. Many physicians have given up the custom of pasteurizing or sterilizing the milk used in infant feeding, especially now that in most cities it is possible to obtain a guaranteed or certified milk of very low bacterial content. While this may be a wise policy during the winter months it is not safe during the summer, no matter what guarantee goes with the milk, and all infant food should be pasteurized or sterilized from the first of June until the first of October in the latitude of New York.

**Treatment.**—The first step in active treatment is to withdraw the food which the infant has been having for twenty-four hours. Next give a purgative that will thoroughly clear the intestinal tract. It may be that this has been already accomplished by the natural process, for the diarrhea in these cases may be regarded as an attempt to get rid of the offending material. If the stomach is not disturbed 1 or 2 teaspoonfuls of castor oil will serve the purpose. If there has been vomiting it is safer to use calomel, giving from 0.006 to 0.0125 gm. ( $\frac{1}{10}$  to  $\frac{1}{8}$  grain) every half-hour or hour until the bowels are freely purged. It is a good plan to follow the calomel in a few hours or on the following morning by a saline, as 1 or 2 teaspoonfuls of a saturated solution of magnesium sulphate given in water.

Water is to be given freely at all times to relieve the thirst. After twenty-four hours' fasting feeding is to be resumed with extreme caution. In the case of a breast-fed infant, nursing may be permitted for five minutes at intervals of four to six hours at first. The duration of the

nursing and its frequency may then be increased according to indications. If return to the breast milk aggravates the diarrhea it will be advisable to feed the infant with whey or barley-water for another twenty-four hours before trying it again, and in case it seems then to prove irritating it may be necessary to give up that breast milk entirely and get a wet-nurse or resort to artificial feeding.

With a bottle-fed infant feeding may be resumed by giving whey or barley-water in quantities much less than the infant has been accustomed to and at longer intervals. After twenty-four hours of such feeding milk may be given again, beginning with a small quantity added to the barley-water or whey, half an ounce of milk in three or four ounces of barley-water or whey, and gradually increasing the quantity of milk and reducing the diluent, until at the end of a week the infant is getting the quantity of milk or milk and cream to which it has been accustomed. Increases should not be made rapidly, and if at any time the diarrhea increases or the stools show more undigested food, the quantity of milk should be reduced again.

Some prefer to use milk alone in the feeding, beginning by using milk diluted with 9 parts of a 4 or 5 per cent. solution of milk-sugar, which would give a milk mixture of 0.4 fat, 4.5-5.5 sugar, and 0.4 proteid. Using 7 parts of the sugar solution would give a mixture of 0.5 fat, 5.5 sugar, and 0.5 proteid. Using 5 parts sugar solution, a mixture of 0.6 fat, 5.6 sugar, and 0.6 proteid. Using 3 parts of the sugar solution, the mixture would contain 1 fat, 6 sugar, and 1 proteid, etc. After reaching this point in progress, one may well use an 8 per cent. cream instead of the milk, and thus double the percentage of fat. As a rule, infants can take twice the percentage of fat that they can of proteid, and the rule holds in these cases. In some instances it may be necessary to keep the fat percentage low for some time. The stools should be carefully watched throughout. The color and consistency should gradually return to normal. The white lumps or masses of casein may be seen for some days in a milk-fed child, but they should steadily lessen in number and size, and the stools become more smooth. If the fat is not digested, it, too, may appear in the stools in masses, which are rather yellow in color, softer than the curds, and dissolve quickly in alcohol or ether.

The medicinal treatment of these cases amounts to very little. If there is much pain or the movements are very frequent, opium may be given—Dover's powder, 0.015 gm. (gr.  $\frac{1}{4}$ ), or paregoric, 0.60 to 1 c.c. (m x-xv), for a one-year-old child. It is best to order the sedative given after each movement of the bowels, so that if the diarrhea is checked the medication will be discontinued. The opium should never be given until the alimentary tract has been thoroughly cleared out. Whatever opium is given should be administered by itself, and not in a complex prescription, so that the quantity of it may be strictly regulated and its administration promptly stopped when it is no longer necessary.

Bismuth is commonly prescribed in these cases and seems to be of advantage. It must be given in relatively large doses, 0.650 gm. (10

grams) or more every two hours. It may be given in powders, each powder being put in a teaspoonful of water or of food. As bismuth is insoluble and very heavy, it is more convenient to administer it in suspension, as in the following prescription:

$\mathcal{R}$ —Bismuthi subnitratii . . . . .	8.0 gm.	(3ij).
Mucilag. acaciae . . . . .	8.0 c.c.	(5j).
Mist. cretae . . . . .	q s, ad 125.0 "	(3iv). M.

Sig.—4 c.c. (one teaspoonful) every two hours.

As the bismuth is insoluble it may be administered freely to infants of any age.

The general hygiene of the child should be regulated. Light and air should be assured. In the summer the infant or child should be in the open air as much as possible. Often a change from the city to the country will marvellously help these cases. In New York, for example, it is found that a single day on one of the Floating Hospitals of St. John's Guild, which take sick children from the tenement districts down the bay, will have a most decided effect in restoring these patients. Care should always be taken to have the diapers promptly changed, when wet or soiled; otherwise the buttocks become reddened and excoriated.

In older children the same general plan is to be followed. After clearing out the bowels and fasting for twenty-four hours, milk and Vichy water may be given in equal parts. If milk is not well borne, broths may be used instead. The strength of the milk allowed is to be gradually increased. Opium may be given on the same indications as above, and bismuth in large doses is useful.

In every instance the effort should be made to discover the cause of the disturbance and correct it, that there may be no return of the trouble. In this regard we must not only examine into the composition of the food, but take into consideration the method of preparation, the care of the bottles and nipples, or any utensil which may possibly contaminate the food. Older children, who are fed at the table, should have the diet regulated.

#### ACUTE ILEOCOLITIS.

Under the heading of Acute Ileocolitis, Enteritis, Enterocolitis, Inflammatory Diarrhea and Dysentery we gather a group of cases which etiologically belong with the acute gastroenteric infections, since they are caused probably by the same infectious agent or agents, but are distinguished from the cases of simple gastroenteric infection, first, pathologically, by the presence of definite and more or less marked inflammatory changes in the intestine, and, second, clinically, by a longer course and a greater mortality. While these are sufficient grounds for the separation of these disorders and their separate description, it is to be understood that the dividing line is not at all definite and that it is often difficult to decide whether a given case should be classed as an infection without definite organic lesions or as an ileocolitis, until the case is concluded, possibly not until we have seen the results of autopsy.



For the most part the etiology of ileocolitis is that of any acute gastro-enteric infection. (See p. 242.) As already noted, these are the cases in which the Shiga bacillus is most regularly found.

**Etiology.**—Ileocolitis occurs both in infants and in children, cases being not uncommon up to the age of five. It occurs at all seasons of the year, but it is much more prevalent in the summer months. It may follow any of the acute infectious diseases, such as measles or pneumonia. It is especially common among the poorly nourished and debilitated children resident in hospitals or asylums or in the tenement districts of our cities. It may follow an attack of cholera infantum, or acute gastric or intestinal indigestion.

**Pathology.**—The inflammatory process affects mainly the colon and the last foot or two of the ileum. The stomach not infrequently shows the changes of catarrhal inflammation, but is most often normal. The upper part of the small intestine is nearly always normal. The changes in the terminal portion of the ileum may be quite as marked as those seen in the colon. The ileocecal valve and adjacent parts usually show the changes to an exaggerated extent. There are three different types of lesions found in these cases: 1. Catarrhal. 2. Ulcerative. 3. Pseudo-membranous.

1. *Catarrhal.*—The gross appearances in this condition are not at all impressive. The mucous membrane of the stomach may be pale or congested, and coated with mucus which is often stained brown from the admixture of blood. In the small intestine we find scattered areas of congestion and perhaps slight swelling at various parts of the gut; these changes may be found even in the upper part. With the congestion there may be a loss of superficial epithelium so that the mucous membrane looks a little granular. The changes are usually more marked near the ileocecal junction. In the colon like conditions prevail. The congestion may be found throughout or only in the lower part, and there is a similar loss of epithelium. The lymphatic tissue throughout is usually swollen; the Peyer's patches may be swollen and congested. Occasionally there is a superficial loss on the surface of the patches, giving them a moth-eaten appearance. In severe grades of this catarrhal process the congestion and swelling of the mucous membrane may be marked and there may be a sense of thickening in the wall of the gut.

Microscopically, these cases show some loss of the superficial epithelium, infiltration of the mucous coat with small round cells, some swelling of the lymphatic structures, injection of the vessels of the mucosa and submucosa, and some slight degeneration in the cells of the tubules. The changes rarely extend deeper than the mucosa, and the muscular and peritoneal coats are normal.

2. *Ulcerative.*—The ulcerative lesions seen in these cases are of two types—follicular and catarrhal. The ulcers are found in the lower ileum and the colon, rarely in the ileum alone, and not infrequently confined to the colon. In the follicular type the ulceration begins in the solitary follicles, which swell, degenerate, liquefy, and are destroyed. In most cases the ulceration is very superficial, producing a slight dimpling in

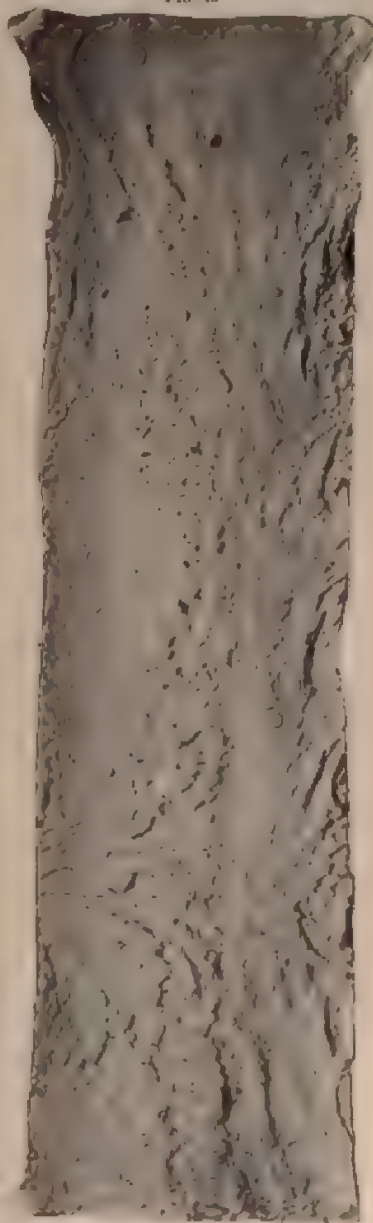


the surface of the gut corresponding to the location of the follicle. The change is more or less general, so that there are numbers of these little dimples especially on the surface of the colon. If the changes are more advanced and the follicles entirely destroyed, deep ulcers with ragged, overhanging edges are produced. These ulcers may extend through the mucosa and submucosa and expose the muscular layer of the wall. Perforation or peritonitis is practically unknown. Microscopically we find the solitary follicles greatly swollen and projecting on the mucous surface, or broken down and undermining to some extent the adjacent mucosa, which may show a considerable infiltration with small round cells.

In the so-called catarrhal ulceration the loss of tissue is more superficial but more extensive than in the follicular ulceration. The destructive process begins about the solitary follicles of the colon, the mucosa being destroyed in a small circular area about them. The fusion of adjacent ulcers may produce a large ulcer with irregular, rounded margins. These large ulcers may extend about the gut, involving a considerable part of the circumference. Such ulcers may be very numerous in the colon; but few are found in the ileum. Microscopically we find the mucosa destroyed in the areas of ulceration and the surrounding tissue infiltrated with small round cells, the infiltration extending in some cases deeply into the submucosa. Associated with either follicular or catarrhal ulceration there may be the general changes of an acute catarrhal inflammation in the mucous membrane of ileum or colon.

3. *Pseudomembranous*.—The pseudomembranous inflammation is also seen mainly in the colon, but affects the lowermost part of the ileum quite regularly. In the colon the whole

FIG. 46



Acute membranous colitis: the surface everywhere roughened by the membranous exudate, the underlying mucous membrane swollen and presenting many minute superficial erosions.

mucosa may be involved or the process may be limited to certain parts of it, particularly the rectum. In this lesion the affected parts are regularly thickened, partly by the exudate, partly by infiltration and edema of the wall. In severe cases the wall may be several times its normal thickness. The most striking feature of the gross specimen is the yellowish or grayish, fibrinous deposit on the surface. The deposit is very rarely continuous over the whole mucosa. Usually there are extensive areas covered with pseudomembrane with intervening areas that are normal or present the appearances of an intense catarrhal inflammation, the mucosa being swollen, intensely red and granular-looking, like raw meat (Fig. 46). There may be minute hemorrhages into the mucosa either in these areas or beneath the pseudomembrane. The membranous deposit is rarely as thick as that seen in croupous inflammation in other parts, such as the pharynx. Usually, it consists of a fine fibrinous layer that is easily washed or brushed off, leaving a deeply injected, red, granular surface beneath. Microscopically the pseudomembrane is seen to consist of fibrin, exfoliated epithelium, leukocytes, and some red cells. The mucosa beneath shows a loss of the superficial epithelium, infiltration of the mucosa and submucosa with small round cells, and edema of the walls. The vessels in the affected areas are deeply injected.

In any of these pathological conditions of the intestinal tract numbers of bacteria can be found on the surface of the mucosa and within its substance. In some instances the bacteria are found in considerable numbers in the submucosa. Some enlargement of the adjacent lymph nodes, the retroperitoneal and especially the mesenteric, is a regular accompaniment of these lesions. The swelling of the lymph nodes is regularly proportionate to the severity of the process in the intestine. On section the lymph nodes may be injected, pinkish in color; more often they are pale. Microscopically the nodes show the changes of acute hyperplasia.

In the lungs there is regularly found hypostatic congestion with scattered areas of collapse. In a large percentage of fatal cases there is a more or less general bronchopneumonia. The kidneys are usually a little swollen and somewhat soft, the cortex pale or injected, and showing moderate cloudiness. Microscopically we find the lesions of acute degeneration. Acute nephritis is much talked of, but very rarely seen. The spleen is usually normal, but may be enlarged and soft. The liver usually shows somewhat more fatty infiltration than is common. I have but once found a true meningitis in association with an ileocolitis.

From a study of thirty-two cases of fatal infection with the Shiga or dysentery bacillus, Howland reports that the pathological lesions may be summarized in four groups: 1. A pseudomembranous inflammation, mainly in the colon, but involving the lower part of the ileum. 2. A hyperplasia of the lymphoid elements in both large and small intestine, in one case in the colon only. The lymph follicles are hyperplastic, the epithelium over them is deficient, and there is some excavation of the follicles themselves, causing "dimpling." 3. A superficial necrosis and ulceration of the mucous membrane not limited to the follicles, and

not accompanied by the formation of pseudomembrane. 4. A group with very few lesions discoverable, macroscopically or microscopically beyond congestion, moderate hyperplasia of the lymphoid tissue, and in one case a little cellular infiltration of the superficial part of the sub-mucosa. There was very slight histological change. The slight changes in this group of cases were mainly attributable to the fact that the cases were mostly terminal infectious in marantic children, whose vital reaction was undoubtedly poor, and in whom the infection ran a very short course. It will be seen that these four groups of cases correspond in a general way closely with the several classes of lesions just described as comprised under the designation "ileocolitis." The lesions of the intestine in children in cases of infection with the Shiga bacillus must, in Howland's opinion, be conceived to be of two kinds: first, those due to the action of the dysentery bacillus itself; second, those due to the action of toxic products and possibly of other micro-organisms.

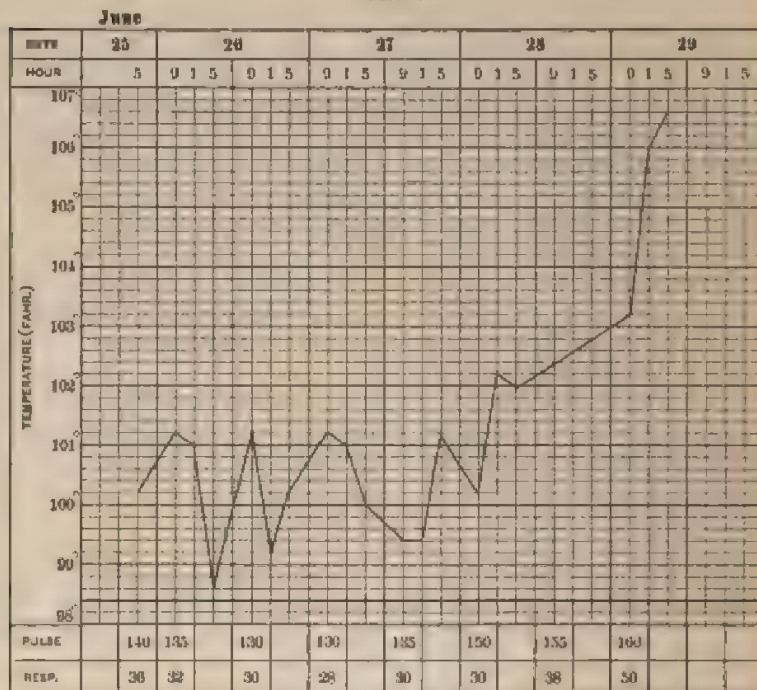
**Symptomatology.**—The mode of onset and the later course of cases of acute ileocolitis varies greatly. It may be a primary affection, or develop secondarily to one or more attacks of acute gastric or intestinal indigestion or gastroenteric infection. In many instances it is a terminal infection in children already exhausted by constitutional disease, rickets, syphilis, or tuberculosis, or by acute disease, such as pneumonia, measles, etc. The cardinal symptoms of an acute ileocolitis in any case are fever, which may be high or low, and diarrhea with the presence of mucus and blood in the stools. We may distinguish several types of the disease of varying severity.

*The Severe Type.*—The onset of the disease is usually sudden, a sharp rise of temperature,  $102^{\circ}$  to  $104^{\circ}$  or  $105^{\circ}$  F., vomiting, rapid pulse, and prostration. The vomiting may be repeated, but is not usually severe. After a few hours diarrhea sets in, first with the passage of the ordinary intestinal contents; then the color of the movements changes to green and they contain undigested food; later they show mucus in considerable amounts and usually more or less blood. The number of stools in twenty-four hours varies greatly, from six or eight to twenty or more. The passage of a stool is accompanied with pain and may be followed by tenesmus. With the full development of the diarrhea and fever nervous symptoms may be marked. The infants or children are restless and fretful. They may be delirious or stupid, or coma or convulsions may occur. The range of temperature is very irregular in these cases. For a few days it is high, reaching  $103^{\circ}$  to  $105^{\circ}$  F.; usually then it takes a lower range, and fever, though present, is not marked, varying from  $99^{\circ}$  to  $101^{\circ}$  or  $102^{\circ}$  F. The pulse remains rapid, the eyes become sunken, the fontanel depressed, and the evidences of exhaustion are marked in the attitude and action of the child. The tongue becomes coated and in the worst cases dry and brown, the lips and teeth may be covered with sordes. The appetite is lost and vomiting may occur frequently. Thirst is usually severe. The diarrhea persists, the movements becoming largely mucous, green or brown in color, with little blood; later in the disease the movements are often foul. Weight and



strength are lost rapidly. At any time the course of the affection may be modified by the development of a bronchopneumonia. After running on in this way for one, two, or three weeks the children die of exhaustion or from pneumonia, or they begin gradually to improve; the fever disappears, the diarrhea lessens, the stools become more fecal, and there may be a slow return to health. In any case convalescence is slow and difficult; the patients suffer from a persistence of the inflammatory conditions in the bowel; any indiscretion or irregularity increases the diarrhea and prostration, and improvement can be secured only by great care and patience. Even after beginning to improve and progressing favorably

FIG. 47



Temperature chart of a case of enterocolitis in a child aged twenty months. Shiga bacillus, alkaline type, isolated from stools.

for several weeks these cases suffer relapses and die of their disease or from some complication, in most instances a bronchopneumonia.

In the most severe of these cases all the symptoms are intense, the fever high, the diarrhea severe; mucus and, it may be, blood abundant; stupor or delirium marked, and exhaustion rapid, so that the children die within a few days of the onset (Fig. 47).

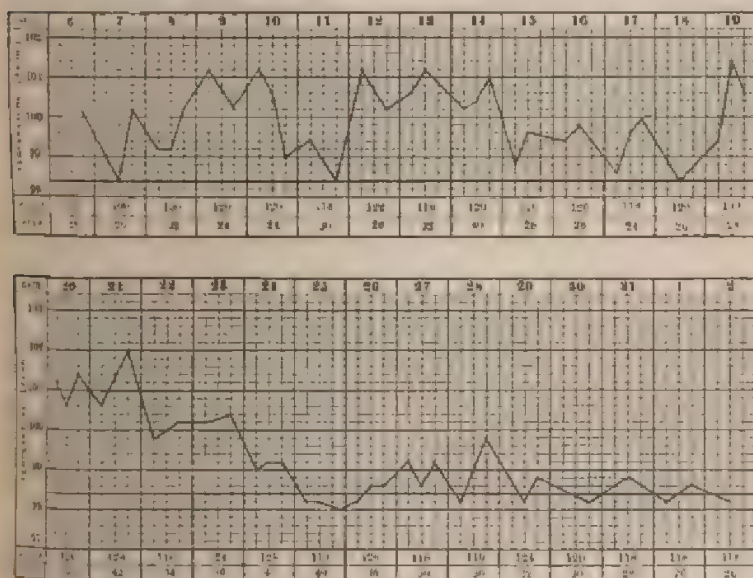
In these severe cases, when fatal, we find at autopsy either an acute catarrhal or pseudomembranous ileocolitis. The recognition of the nature of the pathological lesion before death can be safely based only on one or both of the following points: 1. Prolapse or profusion of the



rectum is very common in these cases and in some cases one may see the prolapsed membrane covered with the characteristic deposit. 2. Shreds of membrane may be found in the stools. If the stools are thoroughly washed, bits of membrane may be floated out in the water, picked out and examined microscopically. If the presence of a pseudomembranous inflammation can be established the outlook is much less favorable than in the acute catarrhal inflammation.

*The Ordinary Type.*—The ordinary type of the disease differs from the severe only in degree. The onset is not so abrupt, the temperature not so high, many cases running their course with temperature not above  $101^{\circ}$  to  $102^{\circ}$  F. (Fig. 48). Vomiting is usually not marked after the onset.

FIG. 48

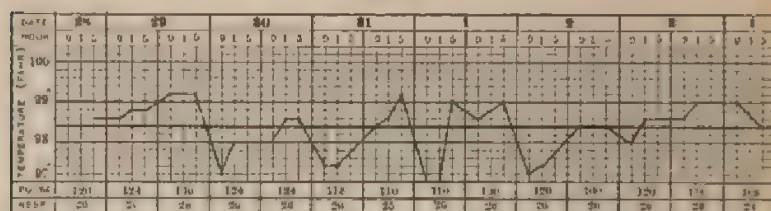


Temperature chart of a case of enterocolitis in a child aged seven months.

The stools are numerous and have the usual characters, the presence of mucus and blood being the essential features. The blood quickly disappears from the stools (usually after three or four days), and these may then resemble those of an ordinary diarrhea. Gripping or tenesmus are usually not marked, but prolapse of the rectum is not uncommon. The prostration in these cases is marked, but not extreme. Weight is lost gradually. The cerebral symptoms are ordinarily slight. The children are fretful and peevish, or may be stupid for some days, but delirium or coma are not seen. Most of these cases at the end of a week show definite signs of improvement and go on to make a good recovery. Convalescence may, however, be interrupted by relapses and in unfavorable conditions the affection may become chronic (Figs. 49 and 50).

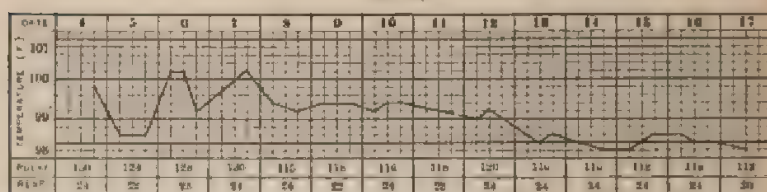
In these cases we are probably dealing with a catarrhal inflammation. If, however, after two or three weeks the diarrhea still persists, with the passage of foul mucous stools, ulceration of the bowels should be suspected. Blood in the stools is not necessarily an indication of ulceration. We have seen that blood is regularly present in the early stages of the disease, when it is due to acute congestion, not to ulceration. It is infrequent, on the other hand, to find abundant ulceration of the bowels in cases in which there has been no blood in the stools. The duration of the disease and the persistence of mucus in considerable quantity in the stools are much more reliable signs.

FIG. 49



Temperature chart of a case of enterocolitis in a child fourteen months old, showing slight fever and subnormal temperatures; recovery.

FIG. 50



Temperature chart of a case of enterocolitis in a child twelve months old. Shiga bacillus, acid type isolated from stools; recovery.

The majority of the cases of an ordinary type recover, but in infants below the age of six months, or in those already weakened by preceding disease, the affection even in a mild form is very fatal. It is very surprising to find how slight the organic lesions may be in the so-called terminal infections.

*The Subacute Type (Follicular Ulceration).*—This type is most often seen in sequence to a number of attacks of acute gastroenteric infection. It may be primary. It is rarely seen under six months of age, but frequent from the sixth month to the end of the second year.

The attack may begin with a sharp rise of temperature and vomiting. More often the onset is insidious and the characteristics of the condition are not shown until the end of a week. In these cases the temperature is but little elevated, but there is some daily fever; usually in the afternoon the temperature reaches 100° to 101° F. Vomiting may occur at the outset of the disease or at rare intervals afterward, but is not a feature

of this condition. Apart from the low fever the stools are most characteristic. These average about six in a day and are green or brown, often foul and full of mucus. Blood may be present in small quantities, never in large amounts. It is absent more often than it is found. The tongue is usually coated, but may be clean. The appetite varies greatly; more commonly it is lost and food is refused completely. The persistent anorexia may be one of the most troublesome features of the complaint. Central symptoms are usually not present. With the low fever and the mucous stools the infant steadily loses weight and wastes until the typical picture of marasmus is developed. The fontanel is depressed, the eyes sunken, the face deeply wrinkled, the skin hanging loosely on the wasted limbs and trunk, and the abdomen either full and tense or depressed and soft; there may be pressure ulcers upon the buttocks, heels, or occiput. Gradually the strength fails, the pulse grows weaker and more rapid, and at last the child fades out of life. Pulmonary symptoms may come on to close the scene, a complicating bronchitis or bronchopneumonia being regularly fatal. The cases usually run three or four weeks. On the other hand, some cases after lingering in a critical condition for weeks gradually begin to regain strength. The stools improve in character and become less frequent; the temperature becomes more even; the child gradually gains strength and may recover. Relapses are especially frequent in this condition, and any one of them may be fatal. Even when recovery is assured the child still shows sensitiveness to any variations in food and diarrhea is easily excited.

The essential features of the condition and the only basis for diagnosis are the low fever and the character of the diarrhea; the stools are frequent, full of mucus, and possibly with a little blood. The course is protracted, usually extending over three or four weeks and sometimes longer. There is no definite limit. The presence of ulcerations undoubtedly prolongs the disease and renders recovery more difficult.

**Nervous Symptoms in the Diarrheal Diseases of Children.**—In any type of these acute diarrheal diseases there may develop very marked and puzzling nervous symptoms. The convulsions of the onset have been spoken of. Later in the course and usually after the subsidence of the high temperature there may develop the condition to which the name hydrocephaloid or spurious hydrocephalus has been given. The fontanel is depressed, the eyes are sunken, the head is drawn back, the pulse is irregular or intermittent, the respiration is irregular and may be Cheyne-Stokes, and the patient is restless and irritable or very stupid, showing no desire for food and rousing only from thirst. The picture is very suggestive of a meningitis. I have seen it made even more so by the presence of strabismus and a slow pulse in addition to the symptoms already named. With improvement in the diarrheal condition these symptoms regularly subside. The explanation of these nervous symptoms has been variously given. It is known that meningitis is extremely rare in these cases, and various other explanations have been offered, such as cerebral anemia or edema, thrombosis of the cerebral sinuses, or uræmia. Of the cerebral conditions named it is only necessary to say



that they are not found with any regularity in association with the symptoms mentioned and are seen in other instances without them, that one cannot accept any of them as a satisfactory explanation. It has been already noted that true nephritis is very rarely seen in these cases; in fact, that we see no more degeneration of the kidney than is seen in any other acute disease. Uremia cannot therefore be regarded as probable. Meningitis is one of the very rarest complications found at autopsy in these diarrheal conditions. I have seen it in but one instance. In any case the question can usually now be settled by the results of a lumbar puncture. In the failure of these various explanations we are for the present compelled to fall back upon the hypothesis that the nervous symptoms are produced by the influence of toxins on the nervous system.

**Diagnosis.**—The diagnosis of an ileocolitis is usually not difficult. Under acute gastric or intestinal indigestion and acute gastroenteric infection it has been pointed out that a number of days' observation may be required to determine the presence of ileocolitis. If in any of these cases the febrile disturbance persists for a week or more, if there is a diarrhea with mucus and blood, particularly the latter, in the stools, we may be quite sure that organic lesions have been established and that the cases may be classed as ileocolitis.

The question of the possibility of typhoid fever occasionally comes up. A continued temperature of the typhoid type is decidedly unusual in these cases. As already noted, after the acute symptoms of the one have passed the range of temperature is distinctly lower and more irregular than is seen in typhoid. Some cases do, however, show a continued fever resembling typhoid, and the abdominal distention and diarrhea add to the resemblance. The absence of any marked enlargement of the spleen, of the characteristic rash, and, finally, of the Widal reaction, enable us to easily exclude typhoid in any suspicious case. It is also to be noted that, except in communities where typhoid fever is rife among adults, it is very rarely met in children under the age of five years, and still more rarely in infants. At the Seaside Hospital, St. John's Guild, where children suffering from diarrheal diseases are sent from New York City, among several hundred cases treated every summer, we find only one or two of typhoid fever. In Philadelphia and Chicago, on the other hand, typhoid is much more frequently seen among infants and children.

The onset of intussusception is often marked by several movements of the bowels containing blood and mucus, and these cases are not infrequently looked upon and treated as ileocolitis. The absence of fever, the severity of the pain, the absence of fecal matter from the stools at the first movement or two, and the presence of an abdominal tumor ought to render differentiation easy, when the possibility of confusion is remembered.

**Prognosis.** This depends upon the age of the patient, his previous general condition, the severity of the attack, and the promptness of proper treatment. In infants under the age of six months, even a mild



attack of ileocolitis may be fatal. In older children who are in good condition and are properly handled, the prognosis is good, unless the onset of the disease be very severe. The pseudomembranous inflammation is very likely to result fatally, even in the strong. In marantic or debilitated children, whatever the age, an attack of ileocolitis is very likely to be fatal. As already pointed out, it is surprising to find how slight the lesions are in many of these terminal infections. Prompt treatment is of importance in any case. Especially in dispensary and hospital practice we see numbers of cases which have been neglected in the early stages and have been allowed to develop a condition (probably of ulceration) from which recovery is either very difficult or impossible, when proper care in the beginning would unquestionably have determined a more satisfactory result. When ulceration has taken place, recovery is not only delayed, but in many cases rendered very doubtful.

**Treatment.** *Prophylaxis.*—This does not differ from the prophylaxis of any of the gastric or enteric infections previously dealt with. The most important point with relation to this particular disorder is to emphasize the necessity and advantage of early treatment. If parents could be taught that any diarrheal disease, in the summer especially, is of great danger and could be induced to put these cases under proper care at the very beginning, the mortality could undoubtedly be greatly reduced.

*General.*—This must be carried out on the lines laid down for acute gastroenteric infections (p. 232).

We begin treatment by thoroughly clearing the stomach and intestinal tract, by washing the stomach and colon with normal salt solution, and giving a purge of calomel or castor oil. We cut off milk feeding until acute symptoms of the onset have subsided and the intestinal tract has had some rest. We then use a small quantity of whey, barley-water, broth, beef-juice, albumen-water, or one of the malted foods, malted or cereal milk, as a substitute for milk. We control the temperature by baths or by washing out the colon. After the subsidence of the acute symptoms we begin the administration of milk in small quantities and in dilute mixtures. We treat the colitis by washing the colon not oftener than once or twice in twenty-four hours, and by astringent injections (Fig. 51). We give stimulants as required. Other medication is limited to the use of some intestinal antiseptic.

*Hygienic.*—The general care of these patients is of more importance than usual, because the disease is likely to be protracted and every factor influencing the general health should be attended to. Removal from the city is of the first importance. The patients should be kept in the country until the summer is over. Return to the city during the summer is regularly followed by relapse. Every summer a number of children just convalescing from an ileocolitis are taken from the Seaside Hospital by impatient parents and brought back to New York. The result is quite regularly a fatal relapse. The daily bathing, prompt change of soiled diapers, rest in bed, and quiet are essential. As Holt says, the cases do better if treated separately than in hospitals. If treated in hospitals, the wards should be small and contain only a few beds.

*Dietetic.*—The dietetic management of these cases presents the most difficult problems in infant feeding. In my opinion, whey is generally better taken and borne than any other substitute for milk. The quantity of feeding and the hours must be regulated, as directed on p. 233, for gastroenteric infection. There is no rule by which we can tell what food is best to give. We may be obliged to try several in succession before we find one that the patient will take. Some infants refuse food altogether, and it is then necessary to feed by gavage. After the acute symptoms of the first few days have passed, milk may be tried in small quan-

FIG. 51



Method of washing out the colon, showing the position of the child and the height of the reservoir.

ties and well diluted. In the cases in which the fever continues high, milk must be withheld until the temperature subsides. If the patient is taking whey, barley-water, or other acceptable diluent for a milk mixture, the milk may be added to this very gradually, a teaspoonful to the feeding in the beginning. If milk alone is tried, it should be given in a mixture containing not more than 2 per cent. fat, 6 per cent. sugar, and 1 per cent. proteid. Peptonization of the milk is often of advantage for a time, but should not be long continued. When milk is once begun, the strength of the mixture must be very slowly increased. Usually a

dextrinized cereal diluent increases the digestibility of milk, and should be tried as the first substitute for peptonization. Relapses are frequent, and require again the administration of calomel or castor oil and return to weaker feeding.

*Local.* As the colon is the part most involved in the inflammatory process, local treatment is of especial value. In the early stages irrigation of the colon should be employed, care being taken that the water is carried as high as possible into the colon and a sufficient quantity of fluid used to thoroughly cleanse it (usually a gallon). Such irrigation, which should be of normal salt solution, 4 gm. to 500 c.c. (1 drachm to the pint), may be repeated twice daily in the beginning, later once a day or once in two days. During the acute stage the water should be used at a temperature of 98° F. Bleeding is rarely sufficient to lead one to hesitate in the use of the irrigation. So long as the reservoir is not more than two or three feet above the level of the patient's body, no danger need be apprehended from the pressure of the irrigating fluid (Fig. 51). During the acute stage the irrigation may be followed by the injection of from 60 to 120 c.c. (2 to 4 ounces) of starch-water containing opium for the relief of pain and tenesmus. For an infant under one year 0.06 c.c. (1 drop) of the tincture, and in the second year 0.12 c.c. (2 drops) of the tincture, may be given every five or six hours in this way to keep the patient comfortable. If the tenesmus is severe 0.015 to 0.030 gm. ( $\frac{1}{4}$  to  $\frac{1}{2}$  grain) of cocaine may be given in a suppository.

After the subsidence of the acute symptoms, astringent enemata may be used. The best are the fluid extract of hamamelis, 4 c.c. (1 drachm) to 500 c.c. (1 pint), or tannic acid, 2 gm. ( $\frac{1}{2}$  drachm) to the pint of water. Such enemata may be used once or twice daily.

*Medicinal.*—It is very doubtful whether antiseptics given by mouth are of any service in this condition, but bismuth or salol are quite regularly given. Bismuth should be given in quantities of at least 8 gm. (2 drachms) in the twenty-four hours. Salol may be given in 0.12 to 0.24 gm. (2 to 4 grains) doses every four hours. (See formulæ, p. 235.)

Stimulants are quite regularly required, and we rely mainly upon alcohol given as directed on page 236.

Opium may be given by mouth for the same indication, and in the same dosage as for gastroenteric infection.

In any case we must rely more upon the general care, diet, and local treatment than the medication for cure.

### CHRONIC ILEOCOLITIS.

Chronic ileocolitis is a common cause of chronic diarrhea in infants, and is seen not infrequently in older children.

*Etiology.*—This condition is regularly the sequel of one or more attacks of acute ileocolitis. The cases are seen most often in the fall among infants or children who have suffered severely from acute ileocolitis but have survived the summer. The etiology of the chronic



affection lies, therefore, in the causes that excite the acute inflammation: bad hygiene, improper food, etc. There can be little doubt that the factors which are operative in producing chronic intestinal indigestion

may also, when long continued, develop a chronic ileocolitis.

Various acute diseases may also be followed by chronic colitis, especially measles, scarlet fever, lobar pneumonia and typhoid fever.

The great majority of the cases must be in the end referred to bad hygiene and bad food. The cases are common under the age of two years; after that age they become steadily less frequent, but are in some instances chronic. Colitis is seen up to the age of ten years.

**Pathology.**—Often the gross appearances of the intestine in these cases is very disappointing. It may look almost, if not quite, normal. The lesions are usually limited to the colon and the adjacent part of the ileum. It is rare for changes to be found in the upper part of the small intestine. The lymphoid tissue of the colon and lower ileum is generally enlarged, and round about the mouths of the solitary follicles in the colon there is some dark pigmentation, while the mucous membrane, as a whole, is of a grayish hue, giving to the surface the "cut-beard" appearance (Fig. 52). The wall of the gut may seem thickened in some cases, thinned in others. There may be ulcers, either of the catarrhal or follicular type, but they are quite infrequent, cases with ulceration usually proving fatal before the condition has become chronic. The catarrhal ulceration is more frequent than the follicular for like reason. In this case the ulcers are very superficial and, as Eustace Smith observes, are best seen by looking obliquely on the surface.

Chronic follicular colitis; solitary follicles enlarged and pigmented, occasional slight excavation of the follicles.

They may be on the summits of the longitudinal folds, when they are long and sinuous, or between them, when they are small and round. In rare instances cysts may be found in the mucous membrane.



Microscopically there is an infiltration of both mucosa and submucosa with small round cells, with destruction of many of the tubules of the mucous membrane due to compression. In long-standing cases there may be a considerable formation of connective tissue. These changes are not continuous, but are scattered in patches through the wall. The mesenteric lymph nodes are swollen and show excessive cell proliferation.

The associated lesions are found most regularly in the lungs, either in hypostatic congestion or consolidation, or as a bronchopneumonia. These changes are regularly found in the lower and posterior parts, the anterior parts being pale and bloodless.

**Symptomatology.** In whatever way established the essential symptom of chronic ileocolitis is diarrhea. In the early stages the stools may resemble those of a chronic intestinal indigestion. They may be abundant, pultaceous, lumpy with a little mucus, or grumous and more like pus. Gradually they lose their consistency, become thinner, more frequent, and contain more mucus and undigested food. The number varies greatly; in some cases not more than five or six a day; in others there may be as many as twenty in twenty-four hours.

The color varies from gray or green to dirty brown. The constant features are the presence of mucus in quantity and undigested food.

The more frequent and watery the stools become, the less apparent are these characters, but they are practically constant. When the stools are few in number it is quite evident that they consist largely of mucus. Blood may be found in the passages, but rarely, even in conditions where ulceration is present. With this chronic diarrhea there are the other symptoms which belong to chronic digestive disturbances. The children are irritable and peevish, as a rule, when they suffer from flatulence and colic; but in other cases where they are free from pain they are singularly placid and listless. Vomiting occurs but rarely. Food is usually taken eagerly. The abdomen may be distended, but is rarely tender, and is often retracted. The walls become thin and may show the veins prominently; but the veins are not dilated. About the genitals and buttocks there may be considerable redness, or even ulcerations from the irritation of the discharges. The other symptoms are those of marasmus or malnutrition. The fontanel, if open, is depressed. The tongue is coated in some cases; in others red and glazed. The skin is usually of a peculiar muddy hue; the mucous membranes are pale and anemic. The facial appearance gives the baby the wizened look of a little old man. Upon the rest of the body the skin hangs in folds with almost no subcutaneous fat and little muscle beneath it. The patients may increase in stature, but do not gain in weight. Eustace Smith states that in this condition dentition may be continued in a normal manner. The temperature is not elevated and is quite regularly subnormal, sometimes falling as low as 95° F. in the mornings. J. Lewis Smith used always to say that such subnormal temperature was a sure sign of approaching demise. In the late stages edema of the hands and feet, gradually becoming more general, may be seen without albuminuria. The urine usually shows no abnormalities of importance. The circulation is poor and the hands

and feet are regularly cold; the pulse is weak. The respiration is feeble and shallow. The eyes are usually clear and bright.

In such condition an infant may linger for weeks or months and then begin to slowly improve. The diarrhea lessens, the stools become more normal in consistency, and the infant begins to show some animation. Eustace Smith makes the curious observation that the return of tears is of favorable significance. The weight may begin to show a gain and very slowly the infant makes progress. In any event the progress is very slow and relapses are frequent. It is generally several years before a child returns to normal and many of these children show the evidences of their loss all through childhood.

In most cases strength is lost gradually until death ensues from exhaustion, or from a complicating bronchitis or bronchopneumonia. The duration of the disease is from two months to a year. Holt says that very few of the cases survive after four months.

**Diagnosis.**—The problem in this relation is to determine whether the intestinal lesions themselves are sufficient to account for the condition or whether there is some underlying constitutional disease. Rickets and syphilis have such characteristic signs that they can be easily recognized and excluded upon the results of the physical examination. The greatest difficulty is to exclude tuberculosis. It has become so common to speak of these cases as consumption of the bowels that a misleading conception of them has become quite general. As a matter of fact very few of these cases are tuberculous, yet from time to time we find tuberculosis present in cases in which it had not been suspected. Tuberculosis is certainly more common in children in hospitals or asylums than in general practice. It is to be considered carefully in cases with a tuberculous family history. The presence of pulmonary consolidation is of some importance. If this involvement is in the posterior and lower parts of the chest, it may be either tuberculous or simple bronchopneumonia. If the consolidation is anterior, it is almost surely tuberculous (Holt). In any case of pulmonary involvement it may be possible by using a cotton swab to secure some of the sputum from the pharynx and determine the presence of tubercle bacilli. In the tuberculous cases the abdomen is more likely to be distended and enlarged mesenteric lymph nodes may be felt. In some instances tubercle bacilli may be found in the mucus of the intestinal discharges. The presence of blood in the stools is rare in any case and is not distinctive.

**Prognosis.**—The age of the patient, the surroundings, and the severity of the diarrhea are the principal factors in determining the prognosis. Infants under the age of six months regularly do badly. The prospect for those in hospitals or asylums is decidedly poorer than for those in private families. Ability to command favorable surroundings, to secure good nursing, and to carry out the various details of care and feeding is of great importance. The severity of the disease depends largely upon the presence or absence of ulceration. There are no decisive symptoms of the presence of ulcers. They are most likely to be present in those who have had repeated attacks of acute ileocolitis. The more protracted

the case and the more severe the diarrhea the greater likelihood that there are ulcers. The older the child the better the prospect of recovery, especially if the conditions are favorable for careful systematic treatment.

**Treatment. Hygienic.**—Fresh air with protection from exposure to cold are of prime importance. During the summer such cases must be kept out of the cities. In the fall or winter the sick-room should be kept as nearly as possible at a temperature of 68° to 70° F. and yet be well ventilated. For this purpose an open fire is particularly desirable. Two rooms should be used in order to secure proper airing and cleaning without exposure to the patient. Great care should be taken in bathing not to chill the patient. In bad cases Eustace Smith advises a bath of one minute in hot soapsuds, or in extreme cases he forbids bathing altogether, except local sponging after each stool. A flannel binder should be worn constantly and the feet protected by woollen stockings. It is necessary to keep the feet warm in all cases, and where this cannot be accomplished otherwise a hot-water bottle should be kept at the feet. Prompt changing and removal of soiled diapers are necessary. The buttocks and genitals should be carefully cleansed after each stool and then thoroughly dusted with a good toilet powder to prevent irritation or ulceration.

**Dietetic.**—Upon the proper management of the diet in these cases the hope of success in treatment mainly depends. The guiding principle is to give adequate nourishment in such form as to leave the smallest possible residue to irritate the inflamed ileum and colon. Farinaceous foods must be cut out altogether or reduced to a minimum. The proportions of both fat and proteid must be greatly reduced to meet the weakened digestive power of the infant. The best materials for food in the early stages are whey, weak veal-broth or chicken-broth, and barley-water, the last being the one form of farinaceous food which seems to be well borne in these cases; it may be dextrinized with advantage. These foods must be given in small quantities, at intervals of not less than two hours. In the first year of life, as a rule, not more than six or seven feedings should be allowed in twenty-four hours; in the second year five feedings are sufficient. At the outset the amount allowed at one feeding should not be more than half of what the age would warrant. Only as the digestion improves should the quantities be increased. Any of the foods recommended for the later stages of acute ileocolitis may be tried.

After a week or two upon these very dilute foods, if improvement has begun, peptonized milk may be tried. The peptonization should be complete at first and the milk may be added in small quantities to the whey or barley-water, or given by itself. The peptonization may then be gradually reduced. In some cases fat cannot be digested and it is necessary to use skimmed milk.

Cereal or malted milk may also be given. Scraped beef is often well borne by patients over a year in age. The reason for employing such a number of foods is that a certain variety is necessary and a mixed diet is found to agree better than a more monotonous one. Thus, as a diet for a nine-months-old infant, beginning to gradually return to milk food,



Eustace Smith recommends the following: *First meal:* One teaspoonful of Mellin's food dissolved in four ounces of sterilized milk and barley water, equal parts. *Second meal:* Four ounces of veal-broth of the strength of a pound of meat to the pint of broth. *Third meal:* Four ounces of whey, containing a dessertspoonful of cream. *Fourth meal:* The unboiled yolk of one egg, plain or beaten up with a tablespoonful of cinnamon-water, a little white sugar, and ten drops of brandy. *Fifth meal:* Same as the first.

When once we have succeeded in getting an infant to digest sufficient food to maintain nourishment, we can usually by gradual increase secure a slow gain. In the early stages we may be satisfied to avoid loss if only we can see a gradual improvement in the stools. Efforts to hasten gain in weight only too often result in overtaxing a weakened digestion and increasing the symptoms.

*Local.*—This should be carried out as directed under acute ileocolitis. The colonic irrigation may be carried out once a day at first; later every other day. The astringent enemata should be of service used in the same way. Pain and tenesmus may require the use of sedative suppositories. Prolapse of the rectum, when it occurs, is produced by the relaxation of the tissues and the straining. The treatment of the diarrhea and the astringent enemata are usually sufficient.

*Medicinal.*—Medicines are of peculiarly little service in this affection. In beginning treatment or with any increase in the symptoms, a full dose of castor oil, 4 c.c. (1 drachm), for a child under a year; 8 c.c. (2 drachms), for one in the second year; or calomel, 0.065 to 0.13 gm. (1 to 2 grains) given in 0.01 to 0.015 gm. ( $\frac{1}{4}$  or  $\frac{1}{2}$  grain) doses hourly, may be given. Excessive peristalsis or pain may be checked by occasional doses of opium, 0.01 to 0.015 gm. ( $\frac{1}{4}$  to  $\frac{1}{2}$  grain) of Dover's powder or 0.60 to 1 c.c. (10 to 15 minims) of paregoric to a child under one year. Antifermentative or antiseptic drugs by mouth have very little effect. Bismuth may be given in the manner described on page 235. It is of no use in small doses, and unless some definite effect can be shown from its use, it had best be omitted.

Stimulants are often required in conditions of exhaustion. Alcohol in some form should be given as described under Acute Ileocolitis. Later, iron and arsenic may be employed. In very wasted children thorough rubbing with some oil, once daily, seems to help them. Cod-liver oil has no special advantage to recommend it for this purpose, and any bland oil may just as well be used, or cocoa butter, such as is regularly employed by masseurs. It is doubtless the rubbing and not the oil that does the good.

#### COLIC.

This scientifically inaccurate and unsatisfactory term serves such a useful purpose in practice and covers so well a multitude of abdominal pains that it maintains its place in our medical books. Under the term "colic" we comprehend any sudden, sharp pain referred to the abdo-



**men.** It has already been mentioned in connection with the section on artificial feeding. Any of the acute inflammatory diseases of stomach, intestines, appendix, or any of the other abdominal viscera may be accompanied by such pain, but in common usage these are not considered under this heading. I restrict its use to the sudden, sharp pains due to disturbed function on the part of the stomach or intestine, and accompanied, as a rule, with flatulence. Even in this more limited signification, colic is a symptom, not a disease, but it often so dominates the scene as to demand consideration by itself. Colic is most frequent in the early weeks of life, when the alimentary tract of the infant is undertaking work which is new and in which it experiences difficulty. In most instances the difficulty lies in the composition of the food; in other cases it seems to be an inherent lack of power in the digestive organs of the infant. Colic may be seen either in nurslings or in the artificially fed, more frequently in the latter. The most reasonable explanation of the occurrence of pain is that, by reason either of the composition of the food or weak digestive power, or both, digestion is imperfect and fermentation occurs with the production of gas and resulting distention and pain; in other cases it may be that there is no gas but a local spasm of the intestine, excited by the presence of an irritant.

In most cases, either in nurslings or in the bottle-fed, colic is produced by an excess of proteids in the food; the excess need not be marked to produce colic in a susceptible child. In some cases excess of sugar, particularly of cane-sugar, may be responsible. Excess of fat rarely causes colic.

Overloading the stomach, feeding too frequently or with great irregularity, giving cold milk and the like, and exposure to cold may in other cases be responsible for the disturbance.

**Symptomatology.**—Usually the symptoms come on within five or ten minutes after a feeding. The baby becomes restless and fretful, then begins to kick uneasily, bends its body forward, and the legs up, and cries vigorously and piteously. The face is at first congested, but in severe attacks it soon becomes pale, with a certain blueness of the lips. The hands and feet are usually colder than normal. The crying continues for a few minutes, or it may be hours; then gas is raised or passed, or the spasm gradually relaxes, the attack passes off. These attacks may be repeated after each feeding, or only occasionally. Infants are peculiarly likely to suffer during the evening and night, and in bad cases the crying is almost incessant. When intestinal distention is the cause of colic the symptoms are often delayed for an hour after feeding.

The colic may be accompanied with other symptoms of an indigestion, frequent vomiting, diarrhea, with stools green and containing mucus, or it may occur in babies apparently well and gaining steadily. In the latter case it is more often associated with constipation, the movements being dry, hard, and lumpy. The attacks of colic usually persist for weeks or months, until improvement in the digestive power or modification of the feeding brings relief. Infants suffering from chronic

gastric or intestinal indigestion frequently have attacks of colic throughout the course of their disease.

**Prophylaxis.**—In this is involved all that pertains to the proper feeding of infants. In the case of nurslings irregularity of feeding in the first few weeks is often the cause of colic. Clock-like regularity should be enjoined. Between nursings the infants should be kept absolutely quiet. Analysis of the breast milk may show irregularities which can be corrected. In artificial feeding we must secure the proper composition of the food, the regular feedings with proper quantities, and the perfect cleanliness of every step of the feeding process.

**Treatment.**—For the milder attacks, peppermint-water, 2 to 4 c.c. (one-half to one drachm), is a household remedy that is often helpful. 0.30 to 0.60 c.c. (five to ten drops) of whiskey or gin in hot water will often suffice if the peppermint does not give relief. Heat to the abdomen and extremities, best secured by letting the baby lie upon a hot-water bottle in the nurse's lap, is most helpful. If these fail, or the attack is severe, relief can most surely and promptly be had by washing out the colon with warm water. Some prefer simply giving an enema of four ounces of warm water, or two ounces of cool water and one-half teaspoonful of glycerin, but the irrigation is more prompt and effective. Care should always be taken to keep the feet warm. If all these measures fail, the pain is probably due to spasmodic action and opium in some form will be required for relief. Paregoric may be given in 0.60 c.c. (ten-drop) doses, repeated in half an hour if necessary. The habitual use of any preparation of opium for the relief of colic should never be advised. Starr recommends bromide and chloral in the following form:

Rx—Potassil bromidl . . . . .	1.0 gin.	(gr. xvi).
Chloral . . . . .	0.5 "	(gr. viii).
Syrup . . . . .	15.0 c.c.	(℥ss).
Aq. menthae pip. . . . .	q. s. ad 50.0 "	(℥ij). —M.

Sig.—4 c.c. (one teaspoonful) for a dose, repeated, if necessary, every hour for three doses.

After the remission of the attack the cause of it should be sought, especially in the feeding, and efforts made to correct any irregularities. It may be necessary to omit one or two feedings and give barley-water. Usually with care the frequency of the attacks can be decidedly lessened, if they cannot be entirely prevented. As the baby grows older and digestive power increases, the frequency of attacks tends naturally to become less.

#### CHRONIC INTESTINAL INDIGESTION.

Chronic intestinal indigestion may be met with at any period of infancy or childhood, but it is particularly common from the sixth month to the end of the second year.

**Etiology.**—The causation of this condition is analogous to that of chronic gastric indigestion. The difficulty may arise from weak intes-

tinal digestive power, which may be either congenital or acquired, or from overtaxing or improperly using a normal intestine. There seems little question that a certain number of infants are born with deficient digestive power, especially with respect to the intestine, but in many other cases the normal digestive power is lowered as the result of constitutional disease, improper care or unhygienic surroundings, overcrowding in tenement houses, bad air, and little sunlight. In any of these ways a child may be rendered unable to digest proper food, and the condition develops into a chronic intestinal disorder, but in the great majority of cases the cause of the disturbance is to be found in improper feeding. It may simply be overfeeding with food of proper composition; much more often it is the composition of the food which is at fault. In the case of breast-fed children the fault most often lies in an excess of proteids and deficiency of fat. In some instances the fat may be in excess. The normal milk-sugar never seems to disturb, even when in excess. In the artificially fed it may be the use of starchy foods, which, being imperfectly digested, undergo fermentation and decomposition in the intestine. Much more often here, also, it is excess in the proteid constituents of the food, especially if cows' milk is being used.

Experience constantly impresses upon us the fact that no matter how modified or manipulated the casein of cows' milk is radically different from the proteid of breast milk, that it cannot be taken in the proportion of the proteid of the latter, and that even in minute amounts its digestion is a very difficult matter for some children.

In any of the ways suggested the tax put upon an infant's digestive powers may be too great and may result in a chronic disturbance of the intestinal functions. In the nature of things the stomach may be involved in the disturbance, but, if so, the gastric symptoms are limited to occasional vomiting. The intestinal disturbance is the main feature of the cases under consideration.

In older children the causation of intestinal indigestion is similar: constitutional disease, bad hygiene, faulty feeding. In these cases it is likely to be an overindulgence in sweets, pastry, richly cooked and poorly prepared foods, etc. In many families, as soon as a child is weaned, it is admitted to the family table and allowed to participate in the common food, what ever that may be. Of these children it is commonly said "They can eat anything."

**Pathology.**—Theoretically these cases would show no organic lesions. Practically infants die with symptoms only of chronic intestinal indigestion, and we find that they show some of the lesions of a chronic colitis, thickening of the wall of the gut, enlargement of the solitary follicles, or pigmentation about them, etc. No definite line exists between the two affections. Doubtless most cases belonging in this category would show no organic lesion.

**Symptomatology.**—The affection usually develops insidiously. It may, however, follow an attack of acute gastric or intestinal indigestion. The most constant symptom is looseness of the bowels. The movements are not very many in the day, five to six, and are passed without straining,



with little or no pain. They consist of undigested food, water, and mucus. The mucus is not in large amounts, except during exacerbations of the affection, and blood is never present. The color of the passages is nearly always green, mingled with which are the white flakes or curds of undigested casein. Fat may appear in the stools in yellowish masses, which are readily soluble in alcohol or ether. The odor of the stools may be unpleasant, but it is not foul. The diarrhea varies from time to time, with periods of improvement, when the stools become nearly normal in number and appearance, or, again, constipation may supervene.

In the bad cases the diarrhea persists, the stools become gradually worse, until they do not differ from those of a chronic colitis. In the milder cases there may be periods of some length when the stools are normal.

Apart from the disturbance of the bowels, the chief symptoms are fretfulness and failure to gain, or actual loss in weight. The infants do not appear to suffer much from pain, but they are restless, peevish, fretful, and sleep badly. They may have attacks of colic.

The crying and fretting of an infant with this disturbance may be a very important matter to a family, permitting very little rest or sleep for anyone, so long as it continues.

Often the early signs of rickets, beading of the ribs, slow closure of the fontanel, and delayed dentition, develop; in other instances these are absent, and the infants increase in stature and cut their teeth in a normal way. The weight, if it increases at all, does so very slowly; often it remains stationary, or there is a loss. With this loss in weight the fat disappears, the abdomen usually becomes retracted, the eyes and fontanel are sunken, the skin hangs loosely in folds, but usually remains clear, the eyes are clear and bright, and, except for the wasting, the infants may not appear sick. The temperature is normal or even below normal, the pulse is usually rapid and weak, the infant is anemic and very languid, and lies almost motionless by the hour. In bad cases the infant gradually develops a marantic condition and dies. In favorable cases the diarrhea gradually lessens, the stools improve, the weight slowly increases, the flabby skin fills out, and the infant gradually becomes normal in appearance. The duration of the disease may be months or even years.

Infants in this condition may present various unusual nervous symptoms. Tetany is not infrequent; retraction of the head, irregularity of the respiration, sudden flushing of the skin in large areas, and urticaria or other eruptions may be seen. The buttocks and genitals may become reddened and inflamed by the discharges from the bowels; bed-sores may develop on the occiput, buttocks, or heels. From time to time these infants have exacerbations of the affection, attacks of severe intestinal colic, perhaps a little fever, and an increase in their diarrhea. It may be very difficult to find a definite cause for these changes in the course of the affection.

While chronic intestinal indigestion is most frequent during infancy, it is not at all uncommon in older children. At this time it may be



natural sequence of the disturbance of the earlier period, or it may be produced by recent irregularities of life and diet. Overfeeding, especially with carbohydrates, indulgence in pastry, candies, etc., are the common causes. The affection is persistent and demands care. Children do not grow out of it without this.

**Symptomatology in Older Children.**—These are very variable, both with respect to the intestine and to the disturbances secondary thereto. The condition of the bowels varies in different patients and in any individual from time to time. Constipation is much more common, the movements being gray or brown, putty-like, and being expelled only with straining. When diarrhea is present the movements are gray or brown, rarely green, watery, and contain undigested food, sometimes mucus. The children are poorly nourished, anemic, small of stature, muscularly weak. The prominence of the head and abdomen in contrast with the thin body is often very striking. The tongue is usually coated, but may be red and glazed. The appetite is lost or is very capricious. The abdomen is often protuberant and tympanitic, but not necessarily so. The face is pale; there are dark rings beneath the eyes; the children are languid, easily exhausted by exertion, peevish, and fretful. Various eccentricities are developed out of the play between the evident physical weakness of the child and the sympathy of indulgent parents. The children usually become thoroughly "spoiled." If without appetite, they will hardly touch food; if hungry, on the other hand, they gratify the craving with whatever the fancy suggests. They are indulged in every way to their harm, and not infrequently both disposition and character are spoiled in the process.

The children often suffer from nervous symptoms of more or less severe type. Headache is most common, and may be of the migraine type. Retraction of the head, tetany, attacks of stupor or unconsciousness, and convulsions may occur. From time to time the condition is made worse by some new indiscretion in diet or life, and the patients have acute attacks of pain, more diarrhea, and possibly fever. Fever may also arise from an autointoxication at any time. Otherwise the temperature is normal or below in conditions of exhaustion. The circulation is regularly poor; the hands and feet are always cold. The urine in these cases is usually loaded with indican, the amount of indican being in proportion to the fermentation going on in the intestinal tract. Lessening of the amount of indican is a valuable guide in the management of these cases. Unless cured by proper treatment these cases drag on for years, finally dying of exhaustion or passing into the condition of continued dyspeptics.

Thomson, of Edinburgh, has recently shown me two cases of chronic diarrhea in young men, in which there had been marked failure of development, the patients being still boys in appearance, small in stature, with beardless, boyish faces, the high-pitched voice, the undeveloped genitals of youth. In addition, there were a peculiar sallow, muddy complexion and marked lassitude. The stools in both cases had been proven to be largely composed of the fat ingested, the diarrhea

being apparently due to a failure of the digestion of fat. It was thought that this failure might be due to a defective pancreatic secretion, and one case had been treated with the pancreatic extract for some time, with the result of an increase of several inches in stature in a few months and marked improvement in all other respects.

**Treatment.**—Whatever the age or condition of the patient, proper treatment involves the regulation of the life, the most favorable arrangements with regard to light, air, exercise, bathing, sleep, etc., that are possible. The matter of clothing is of especial importance, since the circulation is always poor and hands and feet cold. Except in conditions of great exhaustion, these patients should be kept out-of-doors as much as possible. If necessary, they may be sent out in a carriage with hot-water bottles to the feet, but under any circumstances they should get fresh air. If confined to the house, the sick-room should be the best the house affords for light and air; in winter preferably heated by an open fire. The temperature of the room should be kept at 68° to 70° F., as a rule, but if the child cannot be taken out-of-doors, it may be wrapped up well and the windows opened for an hour morning and afternoon. If the child is too weak to warrant that measure, then it must be removed into another warm room for an hour or two daily, while the sick-room is aired and warmed again. The infant or child should have a daily bath at such temperature as is consistent with a good reaction. The utmost care should be given to the child's cleanliness and comfort.

The dietetic management of these cases is the keynote of the treatment. *In Infants:* If breast-fed, the mother's milk should be analyzed. For this purpose the contents of a breast should be taken, the breast being thoroughly emptied, as the composition of the milk is known to vary so in the first and in the latter part of nursing time. Such analysis may be made by the method described by Shaw,<sup>1</sup> or more satisfactorily by an analytical chemist. Analysis of the milk will sometimes show irregularities which can be corrected, or it may be that inquiry as to the mother's life will reveal conditions that should be changed. As a general rule, whatever is best for the mother's health will tend to favor the infant's. If in this way or from the infant's stools we can get a clue to the source of difficulty it may be that modifying the mother's life, as suggested under Gastric Indigestion, may serve to set the matter right. The quantity taken by the child at a nursing must be considered also, and determined by weighing the infant before and after a nursing. By regulating the nursing-time we can cut off any excess that may be shown, although I have found that the amount taken at any one nursing often varies greatly without apparent reason. If the disturbance continues despite these measures, the infant should be weaned or a wet-nurse obtained. It is not wise to allow an infant in whom the signs of intestinal disturbance continue to go on nursing. In breast-fed babies it is best to stop at once the food that is being taken, give 0.065 gm.

<sup>1</sup> Archives of Pediatrics, 1903, vol. xx, p. 578.

(1 **G**rain) of calomel in divided doses, and then put the infant on a modified milk mixture suitable to its age. Thus, in an infant of less than three months, we should use a mixture containing 2 per cent. fat, 6 per cent. sugar, and 0.66 proteid; for a child of three months, a mixture of 2.3 per cent. fat, 6 per cent. sugar, and 1.0 per cent. proteid; for a child of six months, a mixture of 4 per cent. fat, 7 per cent. sugar, and 2.0 per cent. proteid. These mixtures should be given in quantities and at intervals suited to the infant's age. This done, the inspection of the stools will show what part or parts of the food are not digested; these must be accordingly reduced. We should not rest satisfied until the stools are normal. It is quite remarkable on what minute quantities of cows' milk an infant can be nourished if only the milk given is digested. Most often it is the proteids that are at fault, the fat less frequently, and the sugar least of all. It is very rarely, as stated before, that sugar is given in excess; the limit of tolerance is much higher than with either of the other constituents. Usually, we find it necessary to reduce both fats and proteid to a low level, in cases of any length of standing. Some infants, even at six or eight months, will not digest more than 2 per cent. fat and 0.25 per cent. proteid, and yet upon mixtures suited to their digestive abilities will slowly gain. We must not expect to see these infants gain normally for some time. If they can be kept comfortable and a gain of an ounce or two recorded in a week, we may be quite sure that they will in the end do well. As improvement comes, the strength of the milk mixture may be increased very carefully, a fraction of a per cent. at a time. Usually the power to digest fat increases more rapidly than that of proteid. Every increase must be made conditionally. If evidences of undigested food appear in the stools, the food must be reduced to the previous level and kept there for some time. If one can tide a patient through a winter or summer in this way, until a time of the year is reached when he may be safely and comfortably kept out-of-doors for hours at a time, more rapid improvement can be expected. Especial care is necessary through the summers from the great dangers of more serious intestinal disturbance.

In some cases cows' milk is better digested if diluted with some cereal water—barley-water, for example. If cereals are used it is desirable to dextrinize them by the use of one of the diastatic ferments.

In other cases the milk may be peptonized with advantage. If peptonization is tried, it should be prolonged until the peptonization is complete; partial digestion seems to be of no value.

It may be that some patients cannot take milk in any form. Such should be given egg-albumen water, whey, chicken, mutton-broth or beef-broth, and beef-juice for several days or a week, and then the milk tried again in very small quantities.

There is always a temptation in these cases to experiment with one or another of the patent foods, and it must be admitted that in some instances the experiment succeeds, but in most instances it fails. We have no means at present of knowing which cases will do well and which not. Nor is there any one of these foods which can be especially recom-



mended. If such foods are to be used at all, care should be taken to select one that contains no free starch. If we find an infant that has already been put on one of these foods and is comfortable upon it, we shall do well, as a rule, to employ the food as a basis for giving milk, adding the latter in teaspoonful quantities to each feeding at first, and gradually increasing the amount of milk while lessening the quantity of the food until tolerance is established for milk. The solution of the difficulty in nearly all cases is to get the infant to digest an adequate quantity of cows' milk, and by one or other of the methods given above we may hope for success.

Lavage of the colon is useful as part of the treatment. Salt solution, from 1000 to 2000 c.c. (1 to 2 quarts), may be used until the colon is completely cleared of its contents. This lavage is to be used daily, until improvement is begun; then it is to be used only on alternate days.

Medicines are of secondary importance, but seem to be of some value. The following prescription is often of service:

R—Bismuth. subnitrat.	3.0 grm.	(3i).
Elaxir lactopeptin.	8.0 c.c.	(3ij).
Mist. cret.	q. s. ad 120.0 "	(3iv).

Sig. —4-8 c.c. (3j to 3ij) after each feeding.

In older children the treatment is often difficult because the control of the dietary is much harder to establish. The co-operation of the parents must be secured. If not, the child had better be put entirely in the control of a competent nurse, who will carry out orders. All the measures relating to general hygiene are of importance, especially fresh air and exercise without fatigue. In the diet it is essential to exclude the starches, sugars, pastry, hot breads or cake, fruits, and all highly seasoned foods. The diet should be mainly nitrogenous and as bland as possible. Meals should be ordered at regular hours and nothing allowed between them. During the second year five meals a day are sufficient and four for the third or fourth year. Milk should be the chief food at first. In severe cases the milk should be peptonized. If fat cannot be digested, the milk should be skimmed. Kumyss or matzoon may be used as substitutes for milk and are sometimes better borne than milk itself. Junket may be used for the same purpose, or chicken-, mutton- or beef-broth. Meat may be given as scraped meat, or finely cut beef-steak or roast beef. The soft parts of oysters may be given occasionally instead of meat. A good diet is as follows: *Breakfast*, 8 A.M., one or two glasses of milk, with dry toast; a soft-boiled egg may be added every other day. *Luncheon*, 12 M., a teacupful of junket or a cup of broth and one or two Boston crackers. *Dinner*, 3 P.M., a tablespoonful or two of chicken, roast beef or beefsteak with toast or zwieback; the soft parts of six or eight raw oysters may be given as a substitute for the meat, two or three times a week. *Supper*, 7 P.M., a glass or two of milk and toast.

After such a diet has been followed for several weeks, vegetables may be added to the dinner, a tablespoonful of spinach, cauliflower, asparagus tops, or celery, well cooked, being allowed at a time.



If the patients are constipated on such a diet, a tablespoonful of Mellin's food may be added to each glass of milk, or the juice of an orange allowed each morning. If medicines are necessary, Holt especially recommends calomel, a full dose, 0.065 to 0.13 gm. (1 to 2 grains), being given at night, and followed by a saline in the morning. This may be given to any case with advantage every five or six days.

It is doubtful whether any of the antiseptics usually recommended are of service in checking the fermentative processes in the intestine, but some physicians have great faith in them. Salol may be given in 5-grain powders four times a day or the salicylate of soda in the following form:

B—Sodii hypophosphitis <sup>1</sup>	. . . . .	0.75 gm.	(gr. x).
Sodii salicylatis	. . . . .	8.00 "	(3ij).
Aque menthae pip.	. . . . .	120.00 c.c.	(℥iv).—M.

Sig.—3 75 c.c. (3j) in water four times a day, after meals.

It is of the utmost importance with respect to final success that the diet be persisted in for many months. Relapses may be easily caused by any indiscretion in the diet, and the whole process have to be repeated.

### CHRONIC CONSTIPATION.

As with intestinal colic, so with chronic constipation, the condition is often only a symptom, as in rickets or pyloric stenosis, but the symptom is of so much importance as to warrant its separate description and consideration.

**Etiology.**—The causes of chronic constipation are many; it is difficult to classify them all.

1. *Anatomical.*—Undoubtedly the relative length and the many convolutions of the infant's intestine favor constipation, most of all the long sigmoid flexure. The length of this part, its distention and the thinness of its walls are very striking in the infant. It is not very unusual in autopsies on children to see the sigmoid extending over into and filling the right side of the abdomen.

Pyloric stenosis is another anatomical cause for constipation which has of recent years been assuming importance. In this case the constipation is due simply to the limitation of the amount of food passing into the intestine. Bands and adhesion in cases of chronic peritonitis may interfere with normal evacuation of the bowels, but they very rarely come into play in childhood. They are occasionally seen after operation for appendectomy.

2. *Functional.*—Deficiency of the normal secretions of the liver and intestine is sometimes seen, the stools then being gray-colored and hard. Sluggishness of peristalsis from some lack of proper nervous tone is undoubtedly quite regularly a factor in the production of chronic

<sup>1</sup>The sodium hypophosphite is added to prevent the mixture from turning black, as it will do without the trisulphate.

constipation. It is also seen in nervous disorders, such as hydrocephalus, chronic meningitis, and the like.

Inhibition may sometimes come into play in producing constipation; an ulcer of the rectum or hemorrhoids, by reason of fear of pain, may lead a child to restrain the movements of the bowels.

3. *Muscular*.—This is usually spoken of as a muscular atony, a combined weakness and loss of irritability, seen as the result of constitutional disease, such as rickets or malnutrition, or from lack of exercise. The muscles, both voluntary and involuntary, are poorly developed, weak and lacking in tone, and the bowels are deprived of the mechanical support and pressure which they should normally have.

4. *Dietetic*.—The cause of chronic constipation in both infants and children is most often found in some deficiency or irregularity in the food. In breast milk it is most often a deficiency in fat with an excess of proteid; there may be deficiency in both these elements. In artificial feeding it is usually lack of both fat and proteid in the early months. Later, it may be the use of sterilized milk; and in children in the second or third year a too exclusive milk feeding, or lack of the starches and sugars which should be supplied in a mixed diet.

5. *Habits*.—Simple lack of training can cause constipation both in infants and children. In some cases there is coupled with this the habitual use of opium for the relief of colic, or of purgative drugs to relieve the constipation.

**Symptomatology.**—There is considerable variation both in infants and children as to what constitutes normal evacuation of the bowels. During the first year an infant ordinarily has from two to four soft movements daily; in the second year one or two. On the other hand, some infants do perfectly well with but one soft stool daily. If the movements are dry, hard, and passed with effort, the infant is constipated, even if having two or three such passages in a day. In bad cases of constipation an infant or child may go two or three days without a movement.

The symptoms produced by constipation vary greatly. In many instances the infant or child suffers only from the difficulty in evacuating the bowel. Prolapse of the rectum or hemorrhoids may be produced by the straining. Often the infants suffer from colic and flatulence, have little appetite, are restless and fretful, particularly at night, and have occasional attacks of vomiting and fever. It is not uncommon to see a sudden rise of temperature to 102° or 103° F. and vomiting due to constipation. The restlessness and sleeplessness are often marked in both infants and older children. Convulsions may be produced by constipation in susceptible infants.

In older children the symptoms are not so severe as in infants, but these often suffer from colic, disturbance of digestion, headache, restlessness in sleep, languor, and irritability, have a muddy complexion, and show defective nutrition.

The stools are usually small, dry, hard, and lumpy. They may be passed with much straining, and may then be coated with mucus from irritation of the rectum, or blood from hemorrhoids.

**Diagnosis.**—The history is usually perfectly clear, but may be misleading. In any case of doubt the movements should be inspected, and it may be found that, though the bowels are moving daily, the movements are insufficient and of a distinctly constipated character.

**Prognosis.**—The affection is regularly chronic, and may take months or years for relief. If neglected the results may be serious in infants, and convulsions may be produced by an aggravated condition. In older children the headaches, digestive disturbance, and debility produced by constipation may be of grave importance. With proper care every case can be corrected. In certain families the tendency to constipation seems to be hereditary, and great difficulty may be encountered in overcoming it.

**Treatment.** *Hygienic.*—Life in the open air and sunlight are of value in any case, but especially in those with muscular atony. In children old enough to run about, care should be taken to see that they get sufficient exercise. Water should be given freely, especially in summer, even to children at the breast.

*Dietetic.* *In Nurslings.*—If the mother's breast milk is over-rich in proteid and low in fat, efforts must be made by combining diet with exercise to modify the character of the milk. If the breast milk is poor in both fat and proteid, full feeding and rest should bring about improvement.

Where the deficiency is mainly in the fat this may be corrected by giving one to two teaspoonfuls of cream (4 to 5 per cent.) after each feeding. Olive oil may be used for the same purpose. Water must be given to these children, especially in summer; and in an infant over three months of age, oatmeal-water may be used for its laxative effect. In infants over six months of age the juice of half an orange may be given every morning with advantage. If these measures fail we had best resort to suppositories or enemata until such time as the child can be weaned. In aggravated cases artificial feeding should be resorted to if these measures fail to give relief.

*In artificial feeding* it is usually necessary to increase the proportions of both fat and proteid, but the proportions of the food must not be carried far beyond the proportions ordinarily employed. Thus, a child under three months of age is not likely to do well on more than 3 per cent. fat, 6 per cent. sugar, and 1 per cent. proteid; a six-months-old child may be given 4 per cent. fat, 7 per cent. sugar, and 2 per cent. proteid. Raising the proportions much beyond these limits is likely to produce indigestion, but if increases are made gradually this may be avoided. In most cases distinct benefit can be had from the use of oatmeal-water as the diluent of the food.

To infants of six months or over the juice of half an orange may be given the first thing in the morning, and as the age increases the quantity of juice allowed may be increased. The orange-juice is usually enjoyed and is effective. Beef-juice may also be given, a teaspoonful three times a day at first, the quantity being increased if it is well borne. Bovinine and like preparations are also laxative, probably from the glycerine in them.



In the second year, instead of using oatmeal-water as a diluent of the milk, we may employ an oatmeal jelly, adding a tablespoonful to each bottle. Additional cream may also be given, a tablespoonful to each bottle, but it is rarely advisable to raise the percentage of fat above 4. The orange-juice and beef-juice may be used in larger amounts—the juice of a whole orange and from  $\frac{1}{2}$  to 1 ounce of beef-juice. Later, oatmeal- or wheat-porridge may be given with cream. All bread allowed should be made of whole wheat or bran, and butter should be used liberally. Cooked fruits are especially valuable: baked apples, stewed prunes, or figs. Of the latter two fruits only the juice should be allowed at first. Later, the pulp may be given finely mashed. Scraped apple may also be allowed in the latter part of the year. In aggravated cases it may be well to reduce the milk feedings to a minimum and use cream only, giving it upon porridge, mixed with potato or rice, or in soups and broths. From 4 to 8 ounces may be allowed daily. Meat is to be given once a day and green vegetables allowed with it.

*In older children* the same general lines are to be followed. Milk is to be limited or excluded. Fat given freely in cream or butter. Only whole wheat or bran bread used, and abundance of fruit, either raw or stewed, given daily. Water should also be allowed freely. Vichy or Apollinaris water may be preferable to the ordinary supply.

*Local.*—Massage of the abdomen should be employed for ten minutes, once or twice daily, the course of the colon being followed in the movements. Cool sponging of the abdomen, followed by friction with a coarse towel, until the surface is reddened, will also be helpful.

Suppositories are of service, especially in infants, while the other measures mentioned are being put in force, or in case they fail to produce the desired effect. In many instances it seems that the only defect is a lessened irritability of the rectum, and even a slight irritation may be sufficient to produce a movement. For this purpose a cone of oiled paper or a pencil of castile soap is inserted in the rectum and held for two or three minutes. By employing such a measure after the morning bath, even infants in their first months can soon be trained to have a movement at that time. If these means are not sufficient a gluten or glycerin suppository may be used. The glycerin is the more irritating, and, therefore, more effective, but it is always best to employ the mildest measure that accomplishes the purpose. Enemata are still more active. Simple injections of warm water, 60 to 175 c.c. (2 to 6 ounces), may be used. To increase the effect, glycerin may be added, 3.75 to 7.50 c.c. to 60 c.c. (1 or 2 drachms in an ounce) of water.

In cases of fecal impaction 15 c.c. to 30 c.c. ( $\frac{1}{2}$  ounce to 1 ounce) of warm olive oil may be injected and allowed to remain for six hours, then followed by simple enemata of warm water or soapsuds.

*Medicines.*—If the measures outlined above fail, it may be necessary to give medicines by mouth, but their use is objectionable, especially if it is to be continued any length of time, and it is desirable to reduce the use of medicines and stop them as soon as possible. Medicines which will stimulate the flow of bile are indicated when constipation is



accompanied with pale-gray or whitish stools. Calomel in small doses may be given from time to time, but it cannot be kept up. Phosphate of sodium, in doses of from 0.130 to 0.324 gm. (2 to 5 grains), may be given in the food, three times daily, to an infant of six months. It may also be given in the following form:

R—Sodii phosphatis . . . . .	2.0 gm.	(gr. xxx).
Syr. manne . . . . .	75.0 c.c.	(℥jss).
Aq. anisi . . . . .	q. s. ad 100.0 "	(℥ij).—M.

Sig.—4 c.c. (one teaspoonful) three times daily for a child under one year.

The carbonate of magnesium may be given in 0.06 to 0.12 gm. (about 1 or 2 grains) doses in a little milk, or the milk of magnesia in doses of 4 c.c. (1 teaspoonful) to a child under one year.

For systematic use nothing is better than cascara sagrada, either in the fluid extract or an elixir, 0.30 to 0.60 c.c. (5 to 10 drops) of the first, 1.3 c.c. (20 drops) or more of the other. In each case the amount required for a daily movement is to be determined by trial, and the dose regulated accordingly. In most cases the dose can be gradually reduced. For children over six months of age the preparations of malt with cascara are very palatable, from 2 to 4 c.c. ( $\frac{1}{2}$  to 1 teaspoonful) may be given. Maltine and cascara sagrada or Trommer's malt are commonly used.

Treatment may be briefly summarized thus: In any case rely mainly upon diet, hygiene, and massage. In infants, if these fail, use suppositories, the mildest that will be effective, or enemata, if necessary. We may be quite confident that with increase in the strength and variety of the food these measures can be abandoned. Medicines are to be employed only in case of necessity, and are to be discontinued as soon as possible. The dosage required must be found by trial, and effort made to gradually reduce it.

## CHAPTER XIII.

### JAUNDICE—DISEASES OF THE LIVER—INTUSSUSCEPTION—APPENDICITIS—DISEASES OF THE PERITONEUM—INTESTINAL PARASITES.

#### ACUTE GASTRODUODENITIS (CATARRHAL JAUNDICE).

ACUTE Gastroduodenitis or Catarrhal Jaundice is a rare disease during childhood and is almost unknown in infancy. It is assumed that the primary complaint in these cases is a catarrhal inflammation of the stomach and duodenum, resulting in such swelling of the mucous membrane of the duodenum as to obstruct the opening of the bile-duct, or extending into the duct itself and blocking the duct by the swelling of its own lining. The minute size of the common bile-duct in infancy and childhood certainly renders obstruction easy, and if our present views of the causation of catarrhal jaundice are correct, it is difficult to understand why this affection is not much more common in childhood, seeing that catarrhal inflammations of the stomach and intestine are so frequent. There is some ground for the view that catarrhal jaundice is a specific infectious disease.

**Etiology.**—So far as known this is that of any acute gastric catarrh, errors in diet, exposure to cold or wet, etc. It is said to occur particularly after some one of the acute infectious diseases—influenza, malaria, etc.

**Pathology.**—We have no opportunity to examine the viscera in these cases, and all that can be said is that we would expect to find the ordinary evidences of catarrhal inflammation in the stomach and duodenum with sufficient swelling to obstruct the flow of bile at the papilla of Vater.

**Symptomatology.**—The affection begins insidiously with the symptoms of a mild gastric catarrh, a coated tongue, nausea, possibly vomiting, a sense of weight or oppression in the epigastrium, and some depression. There may be tenderness in the epigastrium and some slight enlargement of the liver. On the second or third day the jaundice appears in the conjunctivæ and skin and gradually deepens for a day or two. The tongue becomes more heavily coated, the nausea and possibly vomiting continue, the urine is bronze-tinged with bile and is scanty, the bowels are constipated, and the feces become gray or white in color. There is usually a marked depression, but the severe nervous or cerebral symptoms, associated with the condition of cholemia, are rarely seen. Neither is the slowing of the pulse or respiration, nor the distressing itching of the skin common in childhood. The jaundice reaches its maximum in two or three days and then gradually clears up, all symptoms remitting with it. The duration of the disease may be considered as two weeks,

but the pigmentation of the skin may be perceptible for some time after all symptoms have disappeared.

**Diagnosis.**—The manner of onset and the presence of jaundice are characteristic. Except in early infancy, when jaundice occurs either from congenital obstruction of the bile passages or from the disturbances of portal circulation incident to birth, or the extremely rare Winckel's disease, there is no other common cause of jaundice in childhood. Gallstones are practically unknown, and while the bile-duct has been blocked by *ascaris lumbricoides* or some other foreign body, such occurrences are so rare as to hardly require consideration.

**Treatment.**—This should be directed on the lines of any gastric and intestinal catarrh. After a period of rest for the stomach and intestine the diet should consist first of thin cereal water and meat-broth and later of milk, either plain or diluted with Vichy or carbonated water. When the gastric symptoms have subsided semisolid food may be given, the fats and starches being still restricted. Junket, custards, meat-jellies, and the like may be given, and later sweetbreads, scraped meat, chicken, etc. For the constipation calomel and soda may be given in 0.065–0.130 gm. (1 or 2 grain) doses at night, with a saline in the morning.

Apart from the gastric disturbance the patient suffers from the presence of bile throughout the tissues, and efforts should be made by increasing the flow of urine to more quickly get rid of the offending material. For this purpose an alkaline diuretic such as the following may be of service:

℞—Potassii acetatis,			
Potassii citratis,			
Potassii bicarbonatis . . . . .	ad	80 gm.	(3ij).
Aque . . . . .	q. s. ad	120.0 c.c.	(3iv).—M.

℞—4 c.c. (one teaspoonful) in water t. i. d.

Water should be given in abundance.

### JAUNDICE.

The jaundice produced by gastroduodenal catarrh has already been described. The so-called physiological jaundice of the newborn may be mentioned. This is apparently dependent on the circulatory changes in the liver brought about by the changes in the circulation attendant on birth. Jaundice may also occur in the newborn from congenital obliteration of the bile-ducts. (See page 60.)

In later childhood jaundice from any other cause than gastroduodenal catarrh is extremely rare. It may be associated with cirrhosis, or may be produced by blocking of the ducts by foreign bodies, such as ascarides, or by pressure from without from tumors, such as masses of enlarged lymph nodes. Biliary calculi are, as already stated, almost unknown in childhood. In certain instances jaundice is seen in association with the acute infectious diseases, scarlet fever, pneumonia, or Weil's disease.

**Symptomatology.**—The conjunctivæ and skin are stained yellow or yellowish green. The urine is dark yellow or brownish, with a yellowish foam, and reacts to tests for bile pigments or salts. There is loss of appetite, possibly nausea or vomiting. The tongue is coated white. The bowels are usually constipated with gray or clay-colored stools the odor of which is often very offensive. The pulse and respiration are slow and the temperature may be subnormal. The patient is usually lethargic. There may be troublesome itching of the skin. The liver may be enlarged and sensitive. The course of jaundice depends entirely upon the cause.

**Diagnosis.**—The diagnosis is made on the pigmentation of the conjunctivæ, skin, and urine.

**Treatment.**—The treatment must be adapted to the cause.

#### CONGESTION OF THE LIVER.

Acute congestion of the liver is often spoken of, but of the condition we know practically nothing. Chronic congestion of the liver is produced by any obstruction to the return of blood from the liver to the heart, such as occurs in chronic diseases of the lungs and in cardiac failure from any cause.

The liver of chronic congestion is enlarged, the surface smooth, the cut section is full of blood and presents the characteristic appearance described as "nutmeg." The consistency of the liver may be increased by the presence of more or less cirrhosis in these cases.

**Symptomatology.**—The symptoms are limited to enlargement of the liver with possibly some tenderness of the edge.

**Treatment.**—This must be directed to the cause, which, as is stated above, is in most instances the heart.

#### FATTY LIVER.

In this condition the liver cells are infiltrated with fat. The change is usually more or less general throughout the organ. In the individual cells the amount of fat varies; it may completely occupy the cell body or be limited to minute droplets within it. A certain degree of fatty infiltration is found in nearly all well-nourished infants. It is more marked in children that have suffered from diarrheal diseases or tuberculosis, but it is certainly not observed to any unusual extent in marasmus, as is so often stated in text-books. It may be found to be the explanation of the enlarged liver in rickets, or syphilis. It is not infrequently met with after the acute infectious diseases, but it is very doubtful whether the infectious disease has any relation to the condition of the liver, beyond that of having caused death and thus brought the body to examination.

**Pathology.**—The liver is large, the surface is smooth, paler than normal, or reddish yellow or distinctly yellow. The section has the same color,



and a warmed knife drawn over it will be smeared with oil. Microscopically the cell bodies are found more or less replaced by fat droplets.

**Symptomatology.**—Fatty infiltration is the explanation of 99 per cent. of the so-called enlarged livers met with in infancy and childhood. As already stated, a certain amount of fatty infiltration seems to be normal, and it is likewise normal for the liver to be palpable during infancy and at least the early years of childhood. At this time a liver that is normal may be felt a finger's breadth below the free border of the ribs. Rachitic changes in the thorax often cause a larger surface of the liver to be exposed to palpation. A liver that reaches the level of the umbilicus in a child may be said to be enlarged, but if the fact were appreciated that such increase in size was nearly always produced by simple fatty infiltration of the liver, there would be fewer mistaken diagnoses of cirrhosis, etc. The edge of the fatty liver feels normal; its consistency, as determined by palpation, is normal. There are no other symptoms whatever.

**Treatment.**—Treatment must be limited to that of the underlying condition. If there is no other disease present one may be quite sure that with increase in age the enlargement of the liver will disappear.

#### AMYLOID LIVER.

In childhood amyloid degeneration of the liver is most often seen as a sequel to chronic suppuration in Pott's disease or tuberculous osteitis of other parts; it may also follow syphilis, or chronic empyema, or tuberculosis of the lungs.

The pathogenesis of the condition is the same as in adult life: the formation of a peculiar nitrogenous substance, belonging to the class of albumins, which is deposited in the various tissues, especially in the walls of the bloodvessels.

**Pathology.**—The amyloid liver is usually very large and very heavy, pale gray or grayish red in color, and very tough. The cut surface has a peculiar translucent, glassy appearance, and if a little tincture of iodine be poured over it the amyloid parts are stained a deep mahogany brown. Microscopically the degenerated cells are found especially in the walls of the smaller arteries, but also in the parenchyma. The cells have a peculiar, homogeneous, glassy appearance, and the nuclei may be lost. The liver is never affected alone. Similar changes are found in the spleen, and it may be in the kidney, the intestines, the heart, and bloodvessels generally.

**Symptomatology.**—It can only be said that the organ is notably enlarged, hard, and smooth; the edge is sharp. One cannot distinguish by palpation the amyloid liver from a fatty one. There are no symptoms dependent on the enlarged liver in either case, but in amyloid degeneration we have the symptoms produced by the underlying condition and the widespread character of the process.

The children are usually suffering from prolonged suppuration, with

fever. They are emaciated, the skin is remarkably pale and translucent, the blue veins standing out prominently everywhere. The spleen is enlarged, hard, and with a sharp edge, like the liver. The urine usually shows a large amount of albumin and casts, and there may be a general dropsy.

The condition is practically always fatal. Recovery has been reported to follow the excision of a suppurating joint, but it must be very rare. There may be periods of temporary improvement, but the progress is usually steadily downward.

**Diagnosis.** The diagnosis is founded on the presence of an exciting cause, *e. g.*, syphilis or suppuration, on the coincident and similar enlargement of the spleen, both liver and spleen being notably enlarged, hard, and with sharp edges, and usually upon the presence of large amounts of albumin with casts in the urine.

**Treatment.** In syphilitic cases mercury in such preparation as gray powder and large doses of iodide of potash should be given. In other cases the treatment must be directed to the primary disease.

### CIRRHOSIS OF THE LIVER.

Cirrhosis of the liver in childhood is often spoken of, but very rarely seen. Morse says that it occurs once in 20,000 hospital cases. Hatfield, in 1890, collected 156 cases and Musser, in 1899, 129 more, from literature. In more than a thousand autopsies at the Foundling Hospital, New York, I saw but one cirrhotic liver.

**Etiology.**—Congenital syphilis is the most common cause. Alcohol is responsible for from 10 to 25 per cent. of the cases. Very small amounts of alcohol, if taken regularly, may produce cirrhosis in children. Abnormal fermentation or decomposition in the intestine seems to be a more important factor in childhood than in adult life. Cirrhosis of the liver may be dependent upon chronic venous congestion produced by tuberculosis, adherent pericardium, or acquired heart disease.

Ghose and others have reported many hundreds of cases of cirrhosis of the liver in children in India, the etiology of which is obscure. Congenital obstruction of the bile-ducts may cause cirrhosis very early in life.

**Pathology.**—This does not differ from that observed in adult life. The liver is more often enlarged than small. The distribution of the connective tissue varies considerably. It may be about the lobules, or along the bile-ducts, in patches, or in irregular strands. Atrophic changes in the cells are not marked.

**Symptomatology.**—There are no symptoms peculiar to childhood. The early manifestations consist in disturbances of digestion. Later there is ascites with enlargement of the spleen, and of the superficial veins of the abdomen. Jaundice, if present, is slight. There may be hemorrhages from the nose, stomach, or intestines. The bowels may be constipated, but diarrhea is more common than in adult life.

The course of cirrhosis in childhood is usually rapid, the children dying, as a rule, in a few months after the appearance of ascites. There may, however, be periods of improvement.

A number of cases of the hypertrophic cirrhosis of Hanot have been described in children. The affection is very chronic, lasting several years. The liver is enlarged and hard, but smooth. There may be fever at times. There are attacks of pain referred to the liver. Jaundice is common and often deep. The bowels are constipated but the stools are not clay-colored. The urine may show bile. There is no ascites and no sign of obstruction to the portal circulation. The patients often die of malignant jaundice.

**Prognosis.** Except in the syphilitic form this is always bad. Life may be prolonged by treatment, but the disease is incurable and the end sure.

**Treatment.**—This must be conducted on the lines of cirrhosis in adult life. A milk diet is generally best. In the syphilitic cases mercury internally and mercurial inunctions with large doses of the iodides must be given; and in any doubtful case this treatment should receive trial.

If the ascites is considerable, it is best relieved by paracentesis and the operation should be repeated as often as the fluid reaccumulates. The operation of stitching the omentum so as to establish a collateral circulation, as has been done in adults, might be tried in these cases.

**Rare Affections of the Liver.**—Acute yellow atrophy has been observed during childhood and likewise abscess of the liver, echinococcus cyst, and even malignant tumors, but these conditions are so rare, and the symptoms, so far as known, so much like those of adult life, as to render their separate consideration inadvisable.

### INTUSSUSCEPTION.

In intussusception obstruction of the bowels is produced by the invagination or ensheathing of one segment of the bowel in another, just as one part of a telescope slides into the next. Intussusception is the one form of intestinal obstruction common in infancy. Obstruction from Meckel's diverticulum, from bands or adhesions, occurs but seldom.

**Etiology.**—The affection is found especially in male children, the ratio being about two to one. It is very rarely seen under the age of four months, while the period from the fourth to the twelfth month is that of greatest incidence. Cases are less frequent in the second year and after infancy are quite uncommon.

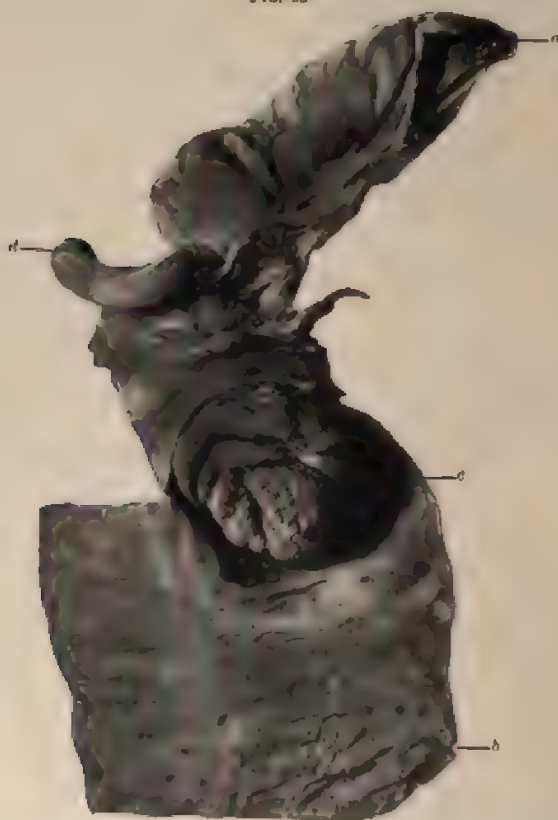
An intussusception being produced by disordered peristalsis in the bowel, any disturbance of the bowel associated with increased peristalsis, as diarrhea, tumor of the intestine, stricture or polypoid growths, the presence in the bowel of irritating food, may be regarded as a predisposing factor. The affection appears in children suffering from such diseases and also in those apparently in perfect health. In the week following



Christmas, 1902, seven cases of intussusception were admitted to the London Hospital. Intussusception is rare, however, among infants or children suffering from the acute diarrheal diseases of summer.

Certain anatomical factors undoubtedly play a part in the incidence of this disease, especially the relative thinness of the walls of the intestine during infancy, and the much greater looseness of both mesentery and mesocolon. The latter especially is notably long, permitting a latitude of motion quite impossible later in life.

FIG. 58



An ileocolic intussusception: *a*, small intestine above; *b*, colon below; *c*, apex of intussusception swollen, congested, and covered with membranous exudate; *d*, appendix.

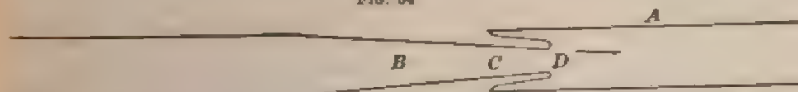
**Pathology.**—An intussusception may involve only the small or large intestine, and is then known as ileal or colic, according to the part affected (Fig. 53). In most cases, however, the intussusception involves both ileum and colon. The conditions can best be illustrated by diagram (Fig. 54).

The outer or ensheathing layer *A* is known as the intussusciens; the inner or ensheathed layer *B* is the intussusceptum. The apex of the



intussusceptum is at *D*, the neck at *C*. In most cases in the beginning the outer sheath is colon, the intussusceptum is small intestine. The apex of the intussusceptum is often the ileocecal valve, and in such case the valve remains the apex of the intussusceptum, the increase coming from infolding first of the cecum and later of the colon. In other cases the ileum slips through the valve, which then forms the neck of the intussusciens and the increase will then be made by more and more of the ileum passing through the valve; the neck remains constant, while the apex continually changes. Numerous cases are on record in which an invaginated Meckel's diverticulum or appendix was found at the apex of the intussusception. The intussusceptum may be only an inch or two or several feet in length. Owing to the fact that the mesentery is carried in with the intussusceptum, the intussusception is often curved in upon itself toward the mesenteric attachment. In most clinical cases intussusceptions are produced by the telescoping of an upper into a lower segment of the gut; in a certain number of cases the process is reversed and a lower portion is invaginated into an upper. Intussusceptions are nearly always single, but double and even triple intussusceptions have been recorded. Multiple intussusceptions are very rarely seen clinically, but are a common occurrence in the post-

FIG. 54



Diagrammatic drawing of an intussusception: *A*, intussusciens; *B*, intussusceptum, *C*, neck; *D*, apex of intussusceptum.

mortem room. It is now well known that intussusception may occur during the final hours of life without giving clinical symptoms. These agonal intussusceptions are all in the small intestine, may be very numerous, as many as a dozen in one case, and may be either of ascending or descending type, or, indeed, of both. There are no pathological changes in the intestine in these cases.

The pathological changes found in clinical cases vary with the length of time which has elapsed from the formation of the intussusception, but still more with the amount of interference with the lumen of the bowel and the nutrition of the parts involved. The intussusceptum becomes deeply engorged with blood and swollen, the enlargement being greatest at the apex, a fact which accounts for the difficulty often met with in reducing the last few inches of the intussusceptum. Following the swelling there may be hemorrhage into the tissues of the intussusceptum, which soon, unless the constriction is relieved, sloughs off, the separation occurring at the neck. Usually after an intussusception has existed for two or three days, there are more or less firm adhesions between the serous surfaces of the intussusciens and intussusceptum. These adhesions may develop very rapidly, or they may be entirely absent after a week. In chronic cases they constitute the greatest obstacle to reduction. At any time a general peritonitis may be excited,

in some cases apparently from infection through the weakened intestinal wall, in others from perforation of the wall of the gut at the neck.

**Symptomatology.**—The onset of intussusception is usually very sudden and acute. An infant apparently in good health is suddenly seized with severe abdominal pain, cries vehemently, flexes the legs on the abdomen, vomits, and is greatly prostrated. The vomiting continues, the vomitus being at first ordinary gastric contents; later it may be bile-tinged and finally it is fecal. With the onset of the attack the bowels move once or twice, the stools consisting of normal feces. Very soon there are movements of blood and mucus, looking very much like currant-jelly. Prostration is usually marked from the beginning. The temperature at the outset and usually for several days thereafter is normal; the respiration is normal, the pulse is rapid and feeble. The facies is usually pale and anxious. The infant may take nourishment greedily, but only to vomit. The severe pains and crying are repeated from time to time. The progress of the case is that of any case of intestinal obstruction, the patient not looking very sick, but steadily losing strength through successive days. The striking features are the vomiting, which is successively food, bile, feces; the repeated attacks of severe pain, with collapse, the passage of blood-stained mucus from the bowel, and finally, the presence of an abdominal tumor. The vomiting is regularly persistent; it may be projectile. At first the vomitus consists of normal gastric contents, later it becomes bilious, and still later it may be fecal. Fecal vomiting occurs in only 15 per cent. of the cases in infants, and is not seen until the third or fourth day. When present, it is of considerable importance from the standpoint of diagnosis.

The tumor felt is formed by the telescoped intestine, and is, therefore, usually in the line of the colon; and as the colon is foreshortened by the process of intussusception, the mass will most often lie near the hepatic flexure or in the position of the transverse colon. The tumor is round and usually long, described as sausage-shaped. It may be felt to harden under the hand. The position and size of the tumor vary from time to time with progress of the intussusception. It is possible for the mass to be felt by the rectum, or in extreme cases it presents at the anus like a prolapse of the rectum. Not infrequently no tumor can be felt. The abdomen is usually soft and may be retracted, yet on account of pain and the natural sensitiveness of an infant to any manipulation it may be impossible to obtain a satisfactory examination without an anesthetic. After the first day or two the abdomen becomes distended and tympanitic. Relaxation of the rectal sphincter has been noted in cases in which the tumor lay in the rectum or sigmoid. After the onset, constipation is usually absolute, neither gas nor feces being passed, but this fact is often overlooked by reason of the passage of blood and mucus. The amount of blood passed is small, usually only enough to tinge the mucus; in some instances the blood is more abundant than the mucus. There may be frequent passages of small amounts of blood and mucus every hour or two.

At the onset the temperature is usually normal. After the first day

or two it may show a rise of one or two degrees, but it is never in the early stages in proportion to the prostration or collapse. Late in the disease the temperature mounts steadily, irrespective of the presence of peritonitis. The latter is comparatively rare and is usually limited to the immediate neighborhood of the intussusception. Rupture of the gut is rare under any conditions.

The urine is usually scanty from the repeated vomiting, but the symptom is of little value.

The course of the disease in infants is nearly always acute. In older children the progress is slower, and in some a condition of chronic intussusception is developed. The affection may be fatal within twenty-four hours, but most of the cases are protracted for four or five days. The duration appears to depend mainly upon the age and resistance of the patient and the site of the obstruction. As a rule, the higher the obstruction the severer the vomiting and prostration, and the earlier the exhaustion. Cases rarely last beyond a week, unless the obstruction is only partial and the condition of a chronic intussusception is developed.

Spontaneous reduction undoubtedly occurs. D'Arcy Power has recently reported two instances of spontaneous reduction. Treves and others consider that many of the attacks of severe colic may be due to small intussusceptions which resolve spontaneously or under the influence of opium. The symptoms of severe colic and intussusception are certainly suggestively similar.

It is possible that the intussusception may become gangrenous, slough off, and recovery occur spontaneously, the outer and inner tubes of the intussusception uniting at the neck, but in an infant such a result is not to be expected. Snow, of Buffalo, has, however, recently reported a case in which a seven months' child suffered from an intussusception for sixteen days, when a piece of gangrenous intestine six inches in length protruded from the rectum, was ligated, and removed, recovery following. In infants spontaneous resolution is more probable than recovery by this process. In older children it may be more frequent.

In the so-called chronic cases, lasting several weeks or months, the symptoms are not at all regular. Usually there is an abdominal tumor which varies its shape and position from time to time. There are attacks of pain and prostration, and the condition of the bowels varies; in some cases there is diarrhea, in other cases alternating diarrhea and constipation. The recognition of the tumor is the important point.

**Diagnosis.**—The sudden onset without fever, the persistent vomiting, the severe pain with symptoms of collapse, the passage of blood and mucus without fecal matter or gas, and finally the presence of a tumor are the diagnostic symptoms. If the possibility of intussusception is borne in mind the diagnosis is usually easy. The most common error is to mistake these cases for ileocolitis or dysentery. In the latter affection fever is present from the beginning, usually in proportion to the severity of the attack; the vomiting is not so persistent; the stools contain more or less fecal matter in addition to blood and mucus, the blood being usually of small amount; the pain is not so severe, and there



is no tumor. The presence of a tumor would at once exclude this condition, but unfortunately a tumor is not always to be felt in intussusception. In over 60 per cent. of Erdmann's 28 cases no tumor could be felt in either the abdomen or rectum. The rectal examination should never be forgotten.

Without tumor the symptoms point only to intestinal obstruction except that the passages of blood and mucus are fairly distinctive and while intestinal obstruction from bands, adhesions, or Meckel's diverticulum does occur in infancy, this condition is exceedingly rare as compared with intussusception.

**Prognosis.**—The prognosis is always very grave, Leichtenstern's statistics showing a mortality of 73 per cent, and Fitz's 69 per cent. The younger the child the graver the prospect; the earlier the diagnosis is made and proper treatment instituted the better the prognosis. Spontaneous reduction is too rare to be depended upon. If the invagination can be reduced by inflation with air or injections of water the case is hopeful, although in these cases the condition occasionally recurs probably from failure to reduce the last few inches of the swollen intussusceptum. After the second or third day reduction by these methods is rather problematical. Within the last few years great progress has been made by treating these cases promptly by laparotomy. The earlier the operation the better the prospect of successful outcome. The chance of recovery by the sloughing of the intussusceptum and spontaneous cure is too small to be considered in infants.

In chronic intussusception also the prospect is very grave. Here operation is essential, yet adhesions will usually have formed so as to prevent reduction and necessitate a resection—always a difficult and dangerous operation in a child.

**Treatment.**—Once the diagnosis is made, the essential thing is the reduction of the intussusception, and the more promptly this is attempted the greater the prospect of success. As preliminary measures, all feeding should be stopped, the stomach may be washed out to check the vomiting, and morphine given hypodermically to relieve pain and quiet peristalsis, 0.0006 gm. (gr.  $\frac{1}{1000}$ ) to a child a year old. For the reduction of the intussusception conservative opinion still advises the use of inflation with atmospheric air or injections of large quantities of water; with either, abdominal taxis should be employed. In any case inflation or injection is allowable as a preliminary treatment if the method does not lead to procrastination in the performance of laparotomy. The procedures are as follows:

**Inflation.**—The child should be placed upon its back on an inclined plane, head downward. The air is best injected through a large catheter (20 French), attached to an ordinary foot-bellows. There is no exact standard for the measurement of the force that is permissible. The air should be slowly injected; the tumor, if present, should be gently manipulated in the direction in which reduction should occur. Danger of injury to the intestine by these manipulations must be admitted, though rupture has been very rarely caused.



**I**nstead of air, Senn advises hydrogen gas and others carbon dioxide, but as the essential thing is promptness air is usually to be preferred.

**R**eduction is often accompanied by a gurgling sound and a sudden disappearance of the tumor under the fingers.

**I**njections of water are made with the child in the position described. A fountain syringe at a height of four or five feet is generally used. The water should be at a temperature of 100° to 105° F. Milk, saline solution, or gruel are advised as being less irritating, but it is best to use water. The injections are made through a large catheter (20 or 25 French) and are to be directed as nearly as may be on the apex of the tumor. The height of the flow may be increased to six or even eight feet, but the danger of rupture is increased by such procedure, and a pressure beyond four or five feet should not be employed in cases of many days' standing. From one to six quarts of fluid may be used, as much as possible being retained by pressing the buttocks together. Taxis should be applied as in the use of inflation.

If either of these methods results in the reduction of the intussusception, the patient should be kept absolutely quiet, feeding for several days kept at a minimum, and morphine or opium given to quiet peristalsis and promote rest. If symptoms return, injection or inflation may be tried a second time; but with a second return of symptoms after relief, or failure to relieve the condition, laparotomy should be immediately performed.

*Laparotomy* is now advocated by surgeons as the proper treatment for all cases. Emphasis is laid upon the brilliant results of immediate operation. It is now generally admitted that infants bear laparotomy much better than was formerly believed. Statistics show that operations on the first or second day are successful in about 50 per cent. of the cases, and individual operators report better results in limited numbers of cases. The essential steps in the operations are the opening of the abdomen over the site of the tumor, if one be present, and the reduction of the intussusception. This is sometimes combined with an effort to shorten the mesentery in the hope of rendering recurrence more difficult. Reduction may be impossible from the presence of adhesions, from too great swelling of the intussusceptum, or may be inadvisable by reason of the condition of the gut. A resection will in that case be necessary. Conditions calling for such complicated operations greatly lessen the chances of recovery.

In chronic intussusception in older children, palliative treatment may be attempted, in the hope that the intussusceptum may slough off and be discharged with resulting natural cure. Even if this occur, there will be later difficulty from cicatricial contracture of the scar and adhesions.

Operation in these cases is difficult and dangerous, because the conditions usually forbid reduction and necessitate resection of the intestine.

**APPENDICITIS.**

Under the caption of appendicitis are now included all the inflammatory processes involving the appendix and cecum, since we are satisfied that in practically all cases the appendix is the part primarily involved.

**Etiology.**—Appendicitis is rare in early childhood and seldom seen in infancy. In the course of more than 1000 autopsies on infants and children under the age of five years at the New York Foundling Hospital, evidence of old inflammation in the appendix was found but twice. There is a curious predisposition on the part of males, boys being the more affected in the proportion of two to one.

Some cause of local irritation may be found in the appendix, a small mass of hardened feces, a seed or fruit-stone, in some instances pins, or other foreign substance.

Blows or injuries to the abdomen are responsible for the production of some few cases, probably by lighting up some old catarrhal or inflammatory process.

Undoubtedly bacteria play a part in the process, especially the colon bacillus. Most cases must be explained upon the basis of a primary irritation by a catarrhal condition or foreign body in an almost closed sac, with the secondary invasion of pathogenic micro-organisms. The affection is seen in close association with acute tonsillitis. Some observers hold that appendicitis belongs in the category of acute infectious diseases, a view for which there is certainly some ground.

**Pathology.**—So far as known, the pathology of appendicitis in children does not differ essentially from the conditions found in adult life, except that the position of the appendix is more variable in the earlier years. I have seen the appendix lying deep in the pelvis with an abscess formed from it, approaching the rectum; in other instances touching the neck of the gall-bladder, and in still others lying well to the left of the umbilicus.

1. *Catarrhal Appendicitis.*—In this condition there is an acute catarrhal inflammation of the mucous membrane of the appendix. The tube is enlarged, its walls slightly infiltrated, the cavity filled and possibly distended with mucus or mucopus. In some instances the lumen of the appendix becomes obliterated at or near its opening into the cecum, and escape of its contents being prevented a cyst is formed which may rupture into the peritoneal cavity.

2. *Ulcerative or Perforating Appendicitis.*—In this form in addition to the changes present in the catarrhal appendicitis we find an ulcerative lesion of the walls. The ulceration may destroy only the mucous membrane or may perforate all the walls of the tube; the perforation is usually near the tip of the appendix and is caused by a twisting or obliteration of the bloodvessel supplying the appendix.

3. *Gangrenous Appendicitis.*—In a certain number of cases, apparently by reason of interference with the circulation of the appendix,

produced by the pathological process and the invasion of virulent bacteria, the whole appendix becomes black and necrotic and sloughs off; in other instances only a part of the organ becomes gangrenous.

With all severe cases of appendicitis there is more or less acute peritonitis. In the simpler cases we find the peritoneum of the appendix and surrounding parts congested and coated with a little fresh fibrin, and there may be some delicate adhesions. In other instances even without perforation there may be a general acute plastic peritonitis. In cases of ulcerative or gangrenous appendicitis we may find a localized peritonitis before the perforation; after perforation there will be either a localized abscess or a general suppurative peritonitis. The factors determining the fate of the peritoneum in these cases seem to be the position of the appendix, which sometimes favors and sometimes prevents the formation of adhesions; the resistance of the tissues, and the virulence of the infecting organisms.

In cases of old appendicitis we find the appendix bound down by adhesions, thickened, and probably containing pus. In some instances we find small abscess cavities closely walled off by firm adhesions.

**Symptomatology.** *Catarrhal Appendicitis.*—A mild attack of appendicitis in a child is shown by a slight rise of temperature ( $100^{\circ}$  to  $101^{\circ}$  F.), vomiting, constipation, pain in the right iliac fossa, and tenderness over the appendix, usually at McBurney's point (Fig. 55). In many cases it may be difficult to satisfy one's self as to the diagnosis, and the presence of an appendicitis can only be suspected. Doubtless, also, many of these mild attacks pass unheeded by children. After a day or two of fever and pain the trouble regularly subsides,

but the patient is very likely to have recurrent attacks. During the attack there may be only a sense of resistance on palpation of the

FIG. 55



Photograph showing McBurney's point. The dot on the right side of the abdomen midway between the umbilicus and the anterosuperior spine of the ilium represents the location of the point.



appendical region, or there may be a definite mass. With the subsidence of the inflammation these local signs entirely disappear.

*Ulcerative or Perforating Appendicitis.*—The onset in these cases presents great variations. In a typical case the disease begins with a rise of temperature— $102^{\circ}$  to  $103^{\circ}$  F.—rarely with a chill, accompanied by nausea, vomiting, constipation, and more or less severe abdominal pain. The pain in the beginning is diffuse, or is referred to the umbilicus. After twenty-four or forty-eight hours it is localized over the appendix. The bowels are usually constipated, but diarrhea may occur. In other instances the onset of the disease is gradual, the temperature is slight, the constitutional disturbance mild, and the evidences of appendicitis are found on the physical examination, a tumor or mass being found in the right iliac fossa with some tenderness over the appendix.

In still other cases the first evidence of the onset of the appendicitis is the development of an acute general peritonitis, with its characteristic vomiting, rapid, small, hard pulse, drawn facies, rigid, tender abdomen, and great prostration.

On the second or third day of an ulcerative appendicitis, beginning either insidiously or with the classic symptoms, the condition is usually characteristic. The patient lies on the back with the knees drawn up and the facies is anxious and distressed. The temperature is  $102^{\circ}$  to  $104^{\circ}$  F.; the pulse is rapid, but otherwise normal; the respiration is normal or is rather shallow and suppressed, the abdomen being held immobile. The tongue is coated; there is nausea and possibly vomiting, either of food or bilious material, and the bowels are quite constipated. On examination we find the abdomen held almost immobile in respiration; it is usually distended and tympanitic; there may be a prominence of the right side. On palpation there is a distinct resistance in the right iliac fossa, or there may be a definite tumor or mass in the fossa. From this point the further course depends upon the progress of the local process. It may resolve; it may go on to the formation of an abscess; it may at any time produce an acute general peritonitis.

*Resolution.*—When this occurs a plastic peritonitis shuts off the inflammatory process in the appendix and the inflammation subsides. The temperature gradually falls, the constitutional symptoms subside, the local induration or tumor diminishes, and at the end of a week or ten days the patient is convalescent. There may be some induration in the appendical region for weeks thereafter.

*Abscess Formation.*—In these cases the appendix ruptures or perforates, but having been previously walled off by a plastic peritonitis, only a localized abscess results. The temperature usually remains elevated, but may fall, the pulse in either case continues rapid, the nausea and vomiting continue, and the constipation persists, while the local signs increase. The pain may increase, but is often surprisingly small after the abscess has formed. The mass in the iliac fossa continues to increase in size, remains tender, and after a day or two fluctuation may be determined. With the formation of the abscess there may be profuse perspiration. The abscess once formed is now regularly recog-



ized and evacuated. In neglected cases it is possible for such an abscess to rupture externally, either in the flank or in the groin. More often the patient dies of sepsis or of an acute general peritonitis from rupture of the abscess into the peritoneum.

*Acute General Peritonitis.* This may arise either from rupture of a previously localized abscess, or from extension of the inflammatory process. The symptoms are characteristic of the condition. (See p. 302.)

The course of an appendicitis, therefore, depends upon the severity of the pathological process and the local conditions favoring or hindering the localization of the inflammation by the formation of adhesions. The mild catarrhal cases run their course in a few days. The severe cases with localized peritonitis may resolve within a week or ten days. The cases with abscess formation usually reach the climax and are opened within from five to seven days; thereafter the symptoms subside and the patients convalesce. The abscess may be slow in forming and operation may be delayed, recovery being correspondingly slow.

The development of an acute general peritonitis is regularly a fatal complication, most of the patients dying within a few days.

**Diagnosis.**—The diagnosis of appendicitis is, as a rule, not difficult. The mild cases may be easily mistaken for attacks of colic or indigestion, if careful examination of the abdomen is not made. Tenderness in the right iliac fossa, rigidity of the right rectus, or tumor in the fossa should cause one to suspect appendicitis, and subsequent observation should determine the question. From intestinal obstruction or intussusception appendicitis differs in the presence of fever from the beginning; in less persistent vomiting, which is never fecal; in more continuous pain and greater tenderness, and more marked rigidity of the abdominal wall; in the shape, location, and feeling of the tumor; and in the absence of the passages of mucus and blood which are characteristic of intussusception. The mass or tumor in intussusception is round or elongated, is in the course of the bowel, may be movable, and often from time to time changes position; it is more likely to be in the position of the transverse or descending colon than in the right iliac fossa; it may sometimes be felt to contract and harden under the fingers. The sensation obtained in palpating a case of appendicitis is more often an indefinite resistance. If a tumor is felt, it lies nearly always in the right iliac fossa, is fixed in position, and only the surface projecting toward the peritoneal cavity can be palpated. Rectal examination may be decisive between the two conditions.

The possibility of mistaking a right-side pneumonia for an acute appendicitis is to be remembered as of great importance. In certain cases of such pneumonia, probably cases complicated by diaphragmatic pleurisy, there is complaint of pain in the appendical region, with tenderness to palpation and rigidity of the right rectus, a group of symptoms very suggestive of appendicitis.

It may be twenty-four hours or longer before the development of characteristic physical signs makes the diagnosis clear. In pneumonia we look for more rapid pulse and respiration and a disturbance of the

pulse-respiration ratio, some movement of the alae nasi, cough, even if slight, some limitation of motion on the affected side, and a more continued and higher fever than belongs to a beginning appendicitis.

Careful and thorough observation should suffice to detect the characteristic physical signs, either of a pneumonia or of the appendicitis. Rectal examination may be of importance here also, by enabling one to locate a definite mass or tumor in the position of the appendix. Occasionally rheumatic children will show rigidity of the muscular walls of the abdomen. Such cases are without temperature, and the symptoms are not persistent.

*The Blood Count in Appendicitis.*—In a broad way the blood count in appendicitis in children has the same characters and the same value as in adult life. In the earliest years (under five years) the interpretation of the blood findings may be rendered less certain by the greater variability of the blood picture and the greater proportion of lymphocytes normally found in the blood during that period; but appendicitis is, happily, rare at that time, and the reported blood counts in appendicitis in children correspond in general with those of later years. The red cells are usually normal in number and appearance; the important changes take place in the leukocytes. There is regularly a leukocytosis, roughly proportionate to the severity of the disease. Thus the mild catarrhal cases may show no increase at all or a leukocytosis of 12,000 to 14,000. A count of 18,000 or more will, in most cases, signify an acute suppurative inflammation with or without spreading peritonitis. While these general statements may be made, one must remember that such important exceptions occur that the blood count alone must not be relied upon to determine the line of action in any individual case. The blood findings must always be taken in conjunction with the other symptoms, especially the temperature and local signs. As Deaver puts it, the changes in the blood should be regarded as simply one of the symptoms of the disease. A single leukocyte count is of much less value than a series. An increasing leukocytosis, whatever the course of the temperature, usually means an advancing process; a falling leukocyte count, on the other hand, regularly indicates a retrogression of the inflammation. It is to be remembered that in some of the worst cases a leukocytosis may not be found, or the increase in number of white cells is slight. The absence of leukocytosis may then be regarded as unfavorable, as it is in pneumonia or diphtheria (Cabot).

The differential leukocyte count may be of considerable help in determining the presence or absence of pus. A percentage of polymorphonuclear leukocytes greater than 80 almost surely indicates a suppurative or gangrenous process, while if the polymorphonuclears are less than 70 per cent. the process is quite surely catarrhal.

Dowd gives the following observations of Sondern: In three children who had gangrenous appendices the polymorphonuclear percentage was 80.8, 85.8, and 95.2, while the number of leukocytes was 7700, 14,000, and 29,800, respectively. In three other patients who recovered without operation the percentage of polymorphonuclears was 63.5, 62, and 68, while the

corresponding leukocyte counts were 25,000, 8800, and 11,700. It would appear from these counts that the significance of the differential count remains the same, whatever the total number of leukocytes. The caution with which the leukocyte count in any individual case must be interpreted is well illustrated by these counts, since in a gangrenous case we find a leukocytosis of only 7700, while a count of 25,000 is recorded in a case recovering without operation and, therefore, presumably catarrhal. The accompanying table shows the results of the blood examination in a variety of cases:

Sex.	Age.	Disease.	White cells.	Remarks.
Boy	8 years	Catarrhal appendicitis.	May 3, 15,000 " 4, 14,000	Recovered without operation.
Boy	11 years	Suppurative appendicitis.	Sept. 22, 20,500 " 23, 33,800 " 24, 32,000	Differential count, Sept. 24— Polynuclears . . . 82.3 Lymphocytes . . . 5.3 Large mononuclears 11.0 Eosinophiles . . . 1.3 100.0
Boy	14 years	Gangrenous appendicitis.	Nov. 11, 16,000 " 12, 15,000	
Girl	10 years	Suppurative appendicitis.	Nov. 20, 18,000	
Boy	13 years	Gangrenous appendicitis, with spreading peritonitis.	Nov. 27, 17,000 " 28, 18,300	
Boy	11 years	Suppurative appendicitis, with abscess.	Dec. 9, 14,000 " 10, 13,900 " 12, 17,900	
Boy	9 years	Gangrenous appendicitis.	Dec. 18, 18,100 " 19, 16,100	
Boy	9 years	Suppurative appendicitis.	Dec. 30, 24,000 Jan. 2, 24,400 " 4, 23,300 " 5, 18,800	
Girl	6 years	Suppurative appendicitis.	Dec. 19, 20,500 " 22, 27,000	Operation December 19.
Girl	12 years	Suppurative appendicitis.	Dec. 30, 18,600 Jan. 3, 12,000	Operation January 6. Appendicitis with abscess.
Boy	11 years	Suppurative appendicitis.	Dec. 18, 7,000 " 19, 15,600	Differential count, Dec. 19— Polynuclears . . . 82.4 Large mononuclears 7.6 Lymphocytes . . . 10.0 100.0 Operation December 20. Appendicitis with large abscess.

In doubtful cases of pus formation in appendicitis it has been found that the determination of the presence of iodophilic granules in the leukocytes is of some value. The presence of numbers of such granules within the leukocytes is regarded as evidence of pus formation, even if temperature, pulse, and leukocyte count be indecisive; and the absence of the iodine reaction is good evidence that no suppuration has occurred. For the details of the method of making the test one must refer to books on clinical diagnosis.



**Prognosis.**—Appendicitis is always a grave affection in a child and the mortality in series of cases already reported has been very high. Earlier diagnosis and more prompt treatment should greatly reduce this. General peritonitis seems to be more frequent among children than in adults. Of 57 cases of appendicitis treated in two years in the Presbyterian Hospital on the service of Dr. McCosh, 7 were under sixteen years of age, 1 only under ten years. Of these 7, 3 had septic peritonitis. Two of the 7 died and 5 recovered. Both fatal cases had general septic peritonitis.

**Treatment.**—Every case of appendicitis, no matter how mild, should be confined to bed. If vomiting is marked, food should be withheld, and in any case only fluids should be allowed during the acute stage. The bowels should be moved once daily by enema. The practice of using saline purgatives in these cases has been abandoned.

For the pain, an ice-bag should be kept over the appendix, or hot fomentations employed, if the cold is objected to. Morphine or opium should be given only in case of severe pain. There is no doubt that the administration of these drugs by numbing the sensibility to pain renders judgment of the condition of the patient more difficult.

For the rest appendicitis is almost entirely a surgical problem. Radical surgeons insist upon the necessity of operating upon every case the moment the diagnosis is made. Many of the mild cases recover within a week without operation, and most parents as well as patients prefer to avoid operation, if it is possible. Undoubtedly, on the other hand, immediate operation lessens the risks from later perforation, and under present conditions the dangers of the operation itself are slight. In the severe cases there is a question between the advisability of immediate operation and of awaiting the formation of an abscess. The tendency at present seems to be to operate at once if the disease has not existed more than forty-eight hours. After that time it is advisable to delay operation until the abscess has formed and become easily accessible.

In cases of recurrent appendicitis, operation in the interval has come to be recognized as presenting little danger and the best prospect of recovery.

In almost every case of appendicitis there are surgical problems which require trained judgment and skill, and therefore surgical advice should be regularly sought and the physician should welcome the surgeon's aid, even if operation is not imperative.

#### ACUTE PERITONITIS.

Acute inflammation of the peritoneum is a relatively rare occurrence in childhood, but may be met with at any age. It is seen in the fetus and is much more common in the newborn than in the later periods.

**Etiology.**—Acute peritonitis is regularly a secondary process, although a certain number of cases do arise in which it is difficult or impossible to demonstrate the primary factor. In the newborn acute peritonitis



Generally secondary to some infection of the umbilicus, suppuration of the umbilicus or in its vessels, erysipelas of the umbilicus, etc. In rare instances it is secondary to some congenital malformation, such as atresia or occlusion of the rectum. Syphilis is also given as a cause of acute peritonitis in the newborn. The dangers of the early weeks passed, infants very rarely suffer from acute peritonitis. It is then most often secondary to inflammation of the lung, pleura or pericardium; it is not uncommon to see it as part of a general infection of the serous membranes, the meninges, pleura, and pericardium. It may be secondary to the acute infectious diseases: typhoid, dysentery, erysipelas, scarlet fever. It may follow severe inflammation of the intestine, but is surprisingly rare in ileocolitis, etc. It develops in the course of intussusception, strangulated hernia, or ulcerative processes in the stomach or intestine with perforation. A perforating gastric ulcer in childhood is almost unknown. The deep ulcers of the intestine, typhoid, tuberculous, or dysenteric, very rarely perforate; more often the seat of the ulceration is shut off by a local peritonitis. Just as appendicitis becomes more frequent with each year in childhood, it becomes more often the cause of acute peritonitis. The appendix is certainly the origin of most cases arising without apparent cause. Acute peritonitis may follow acute inflammatory or suppurative processes in any of the viscera: liver, spleen, kidneys, uterus, and tubes. Abscesses in the viscera or arising from the spinal column or pelvic bones in Pott's disease, etc., may rupture into the peritoneum and set up acute peritonitis.

Gonorrheal vulvovaginitis may lead to acute peritonitis; gonorrheal infection in boys does not do so.

In a certain number of cases the cause of the peritonitis not being discoverable, we have attempted to cover our ignorance under the designation of "rheumatic" peritonitis.

**Pathology.**—Bacteriologically, we find the staphylococcus, streptococcus, pneumococcus, or the colon bacillus in most of these cases. The colon bacillus will, of course, be found especially with perforation, appendicitis, etc. The pneumococcus is frequently found in cases secondary to pneumonia, pleurisy, or pericarditis, and also in a certain number of apparently primary or "rheumatic" cases. The gonococcus is found in the peritonitis secondary to gonorrhea in the female.

**Lesions.**—In the earliest stage the peritoneum loses its clear, shiny appearance and becomes slightly reddened and hazy. If the process continues, there is an effusion of fibrin alone, or fibrin and serum, or pus. In the fibrinous cases there is a plastic deposit over both the parietal and visceral peritoneum, gluing the coils of intestine and all the opposing surfaces together. Usually these adhesions are very delicate and easily separated, but in old cases they become quite firm. The changes of acute peritonitis may be circumscribed, but in children they are very likely to be general. The serous effusion is rarely large, the serum is found filling the pelvis and the flanks. It may be clear, but is usually cloudy from admixture with fibrin. Pus is most often found in cases of appendicitis or perforation. In these cases the pus has a

very characteristic, foul, fecal odor. The amount of pus may be small or large. It collects as does the serum especially in the pelvis and flanks. The collections of pus may be encapsulated in any part of the abdomen, forming localized abscesses which may perforate through the rectum, bladder, vagina, or even the abdominal wall.

**Symptomatology.**—In infants the symptoms of acute general peritonitis are very indefinite and uncertain. Time and again it appears at autopsy, when it has not been suspected during life. The possibility of the onset of peritonitis must be borne in mind in every case of infection about the navel, likewise the frequency with which peritonitis follows pneumonia or pleurisy. In older children the relationship to appendicitis should be remembered.

The onset of an acute peritonitis in a child is frequently obscured by the preceding affection such as pneumonia, pleurisy, or appendicitis. If there has been no fever previously observed, it now appears, or if present before, it is increased. The temperature is usually high, 103° to 104° F., or more, but a lower range does not exclude the presence of a general peritonitis. Chills may occur at the onset or at any time during the course. The patient from the beginning looks and acts very sick, the eyes appear sunken, and symptoms of collapse—pallor, small, feeble pulse, cold extremities—appear early. The respiration is rapid and shallow, and almost wholly costal, the movements of the diaphragm being inhibited on account of pain. The pulse is rapid, small, and hard. The skin of the body is hot and dry, while the extremities are cold and often somewhat cyanotic.

There is nausea from the beginning and vomiting usually follows and continues steadily to the end. Vomiting may, however, be almost absent in infants. The vomitus after the evacuation of the stomach contents consists of mucus and watery, bile-tinged fluid. The urine is diminished in amount, dark in color, and contains indican in abundance. There may be difficulty in urination on account of pain excited by the movement of the abdominal muscles; there may be retention, or the urine may be passed frequently in small quantities. The condition of the bowels varies. The obstinate constipation of adults is not so frequent in children, the bowels may move normally or there may even be a diarrhea. The local signs are of quite as much importance as the constitutional symptoms. Infants usually lie flat on the back with the limbs straight. In children we may see the characteristic attitude with the knees drawn up. The abdomen is usually full, tense, tympanitic, and very tender to touch. There is a marked rigidity of all the abdominal muscles. The pain and tenderness may be so great that even the slightest motion, or touch, or the weight of the bed-clothes excites pain and causes the child to cry out. The tension of the abdomen may be visible; it is usually better appreciated by touch. The muscular rigidity is a sign of considerable importance. It is very rare that the effusion of serum or pus reaches a sufficient amount to give dulness in the flanks. If the exudate is encapsulated, it gives rise to a localized dulness which should be sought for by light percussion.

The course of an acute peritonitis varies greatly with the age of the patient and the condition underlying it. The onset is often violent, the fever high, vomiting or hiccough persistent, the collapse marked from the beginning, and death ensues in from twelve to forty-eight hours. Especially in the newborn is the rapid course seen, and all the characteristic symptoms may be wanting. The cases of perforative peritonitis are also very swiftly fatal, as a rule. Acute peritonitis, non-suppurative in older children, runs a more favorable course, many of the children recovering after one or two weeks, with a gradual subsidence of the symptoms. The gonococcal peritonitis usually runs a favorable course.

In cases in which the process becomes localized, the temperature may assume a hectic type, an abscess may form, and the process continue for weeks, until the abscess ruptures or is opened.

**Diagnosis.**—The diagnosis of acute general peritonitis must rest in most cases upon the combination of the constitutional and local symptoms, aided in many cases by the previous existence of some source of infection, such as an inflamed navel or an appendicitis. With a typical case the diagnosis is easily made, but as already noted the characteristic symptoms are often wanting, especially in infants, and the disease is overlooked. The presence of abdominal distention with tympany, acute pain on the slightest touch, rigidity of the abdominal muscles, absence of respiratory movement, and the constitutional evidences of severe illness should enable one to make a diagnosis, but oftentimes the abdominal examination elicits only doubtful or uncertain signs, and it is difficult or impossible to reach a conclusion.

**Prognosis.**—Acute peritonitis is always a grave and generally a fatal affection in childhood. All cases in infancy are fatal. All the cases depending upon perforation in older children are fatal. Acute general peritonitis following appendicitis is regularly fatal. Of recent years prompt operation has saved a number of such cases. The so-called primary cases usually run a more favorable course, as does also the gonococcal peritonitis.

**Treatment.**—While the treatment of these cases is essentially surgical, some suggestions as to medical care are necessary when surgical intervention is not possible. The patient is to be kept as nearly as possible absolutely quiet in bed. At the onset the bowels may be freely moved by salines, later no purgative should be given by mouth. Cold should be applied to the abdomen by the Leiter coil, and care taken to keep the flow constant. Many children will not, however, endure the application of cold, and heat must be employed to relieve pain. This can best be done by spongiopiline or flannel wrung out of hot water, the application to be frequently renewed to get the best effect of the heat. A few drops of turpentine may be sprinkled on the cloth, to increase the counterirritation. It is very doubtful whether these measures do more than relieve pain, but they are useful for that purpose. Morphine is to be used hypodermically for the same purpose and to quiet peristalsis. For a child of five years 0.003 gm. (a twentieth of a grain) may



be given as the initial dose, and this amount repeated every two or three hours. The dose must, of course, be regulated by its effect upon the patient. The hypodermic use of morphine is certainly preferable to the administration of opium by the mouth or rectum, but in some cases the latter method may be necessary. For a child of five years we may then begin with three drops of laudanum, and repeat it as necessary. The constipation produced by the use of the opium or morphine is unfavorable and no effort need be made to move the bowels for as long as a week. Then enemata should be employed. Schreiber records a case in which a child suffering from peritonitis and under the opium treatment went twenty-two days without a movement of the bowels without harm.

The feeding of the patients is important. In the early stages with much vomiting nothing should be given by mouth. Any medication necessary should be given hypodermically or by the rectum. The stomach may be washed out with advantage, if the vomiting is severe. After forty-eight hours, feeding may be tried. Peptonized milk, matzoh kumyss, and beef-juice or other concentrated and easily digested food may be employed. The food must be given in small quantities and oftener than once in two hours. Attempts to press feeding will only result in increased vomiting.

For the relief of the distressing thirst small bits of ice may be given to be held in the mouth; the ice also serves to allay the irritability of the stomach and relieve either vomiting or hiccough. If the temperature is very high, cool sponging may be employed to the chest and limbs.

Stimulants are required for the failing pulse. Champagne is usually better borne than any other form of alcohol. A good whiskey is better than a poor brandy. Strychnine, camphor, or whiskey may be used hypodermically.

As the patient shows improvement the opium is to be gradually withdrawn.

In cases of perforative peritonitis the only hope of the patient lies in early surgical interference.

With improvement of technique in recent years much better results than were formerly known have been obtained by laparotomy in these cases. Whenever pus has formed, operative treatment is imperative.

#### CHRONIC PERITONITIS.

**Etiology.**—The occurrence of a primary chronic peritonitis is tuberculous was until recently doubted, and even now some maintain that this process is always tuberculous.

The work of Galvagni, Vierordt, Henoeh and others has, however, established the existence of an independent chronic peritonitis. It is very rare. It occurs mostly in children from six to twelve years of age. The causation is very obscure. Exposure to cold and wet, injury, rheumatism, or measles has preceded the onset.



**Pathology.**—Very few autopsies have been recorded. In a case of Henoch about 500 c.c. of turbid fluid were found in the abdomen, with many adhesions between the loops of small intestine and an enormous, fibrous thickening of the peritoneum in general. There were no traces of tuberculosis.

**Symptomatology.**—The affection develops very insidiously. There may be slight digestive disturbances. The chief and frequently the only sign of the disease is a gradually increasing ascites, which gives the usual physical signs. Usually the effusion is serous, sometimes serofibrinous; the fluid may have a greenish color. In the serofibrinous cases there may be a little evening temperature and nodular masses may be felt in the abdomen. There may be anemia and some loss of weight, but, as a rule, the general health is not markedly affected. After weeks or months the fluid is gradually absorbed and there is a complete return to health.

**Diagnosis.**—The important point is to distinguish this affection from tuberculous peritonitis. The points in differential diagnosis are given under the latter subject. (See page 373.)

**Treatment.**—This must be conducted on the lines of the medicinal treatment of tuberculous peritonitis.

### ASCITES.

By ascites we understand a collection of serum in the peritoneal cavity. It is a symptom and not a disease. Ascites may arise in any form of chronic peritonitis, simple or tuberculous. It may be produced by obstruction to the portal circulation, by cirrhosis of the liver, by tumors, such as masses of enlarged lymph nodes, pressing upon the portal vein; by obstruction to the circulation in the lungs from chronic pneumonia; by cardiac failure of any kind. It may be part of a general anasarca, such as occurs in chronic nephritis, severe anemia, or cachexia.

**Symptomatology.**—The physical signs of ascites are distinctive. The abdomen is distended, tense, and symmetrical. If the patient lies upon the back, there is resonance over the central parts of the abdomen with dullness in the flanks. If the patient turns on one side, the fluid sinks to the other side, with the line dullness rising higher on that side and resonance in the uppermost side. If the patient sits the dullness is in the lower parts of the abdomen and the upper parts are resonant. With the patient lying upon the back tapping one side of the abdomen with the fingers gives rise to a fluid wave which can be felt by the fingers of the other hand placed on the other side of the abdomen. A similar wave may be obtained in a tympanitic abdomen, but if an assistant places the ulnar border of one hand on the linea alba and presses firmly downward the wave in a tympanitic abdomen is cut off, while that transmitted through fluid is not interrupted.

A considerable collection of fluid in the abdomen displaces both liver and heart upward and may give rise to dyspnea or considerably embarrass the heart action.

The fluid in ascites is alkaline in reaction, usually clear, and of light-yellow color. The specific gravity varies from 1005 to 1020, averaging 1010. The albumin content is generally not over 1 to 2 per cent. In children a very few cases of chylous ascites have been reported, the fluid having a milky-white color from admixture with fat. Fat is supposed to occur in the ascitic fluid by reason of some pressure upon the lacteal system, but this has not been satisfactorily demonstrated in cases in which examination has been possible. Fatty degeneration of the cellular elements of the fluid has also been suggested as an explanation, but this does not seem sufficient to account for the quantity of fat present in these conditions. The pressure of a chylous ascites adds to the gravity of the condition; one case of recovery has, however, been reported.

**Treatment.** The treatment of ascites is that of the underlying condition. Whenever the quantity of fluid is sufficient to give rise to much pressure or interfere with the action of the heart or lungs it should be removed by paracentesis.

#### PROCTITIS.

Inflammation of the rectum occurs to some extent in nearly all cases of marked inflammatory processes in the colon or sigmoid flexure. It may, however, occur independently, and in this form merits separate consideration.

**Etiology.**—Proctitis may be produced either by a local irritation, infection or a combination of these two factors. The presence of threadworms, traumatism in the use of suppositories or injections, or the use of irritating materials in either of these forms may excite a proctitis. Infection may be conveyed in cases of gonorrhea or syphilis, or from the use of infected instruments, such as rectal tubes or douche nozzle.

**Pathology.** There may be a simple catarrhal inflammation, with redness, swelling, and increased secretion of mucus; pseudomembranous inflammation with conditions similar to those seen in pseudomembranous ileocolitis; or ulceration, which may be the multiple follicular type seen in the colon, or single, larger ulcers. The large ulcers may be produced by the fusion of a number of small follicular ulcers, or may be simply catarrhal, or in rare cases tuberculous.

**Symptomatology.** Inflammation of the rectum alone produces a fairly characteristic picture. There are frequent movements of the bowels, perhaps as many as fifteen or twenty a day, each movement being accompanied with straining and fretfulness or crying, indicating pain. The reflex action of the rectum may be so increased that the movements are projectile and expelled suddenly. The movements for the most part consist of mucus alone, or, if ulcers be present, mingled with blood. They are followed by tenesmus. In the pseudomembranous cases bits of the pseudomembrane may be found in the stools by washing the mass as in pseudomembranous ileocolitis. Three or four times daily there is

occur freemovements of the bowels, which are yellow and almost normal. The annoyance of the frequent movements, together with the straining and pain, may produce wasting, pallor, and prostration. In some cases prolapse of the rectum may be caused, and in these cases the character of the inflammatory process may be observed in the prolapsed portion of the bowel. In other cases inspection through a speculum discloses catarrhal or pseudomembranous inflammation, with or without ulceration. There is regularly more or less excoriation about the anus from the irritation of the frequent passages.

**Diagnosis.**—The condition is most often confused with ileocolitis. The diagnosis should be made on the observation of the occurrence of some fairly normal movements, while the other passages consist almost wholly of mucus or mucus and blood, which are evacuated suddenly with straining and pain. Inspection of the rectum shows the local inflammation.

**Treatment.**—If a direct cause of the inflammation can be found, such as the presence of thread-worms or the use of irritating suppositories, the removal of the cause may be all that is required. Ulcers will require covering with carbolic acid or a silver-nitrate point.

Most cases require the use of cleansing injections and salt solution, 1 gm. to 500 c.c. (1 drachm to 1 pint), or a saturated solution of boric acid. These injections should be given warm and a sufficient quantity, 50 c.c. (1 pint), employed to thoroughly cleanse the rectum. After these cleansing injections Starr recommends the introduction of 8 gm. (2 drachms) of olive oil or equal parts of olive oil and lime-water. Such injections should be employed twice daily at first, later once a day.

In more obstinate cases after cleansing the rectum with plain water an astringent injection of tannin, 1 gm. to 50 c.c. (10 grains to the ounce), or nitrate of silver, 0.1 gm. to 50 c.c. (1 grain to the ounce), should be introduced, and after five or ten minutes the excess washed out either with plain water or the salt solution.

Such astringent injections are to be repeated at intervals of two or three days, until improvement is noted; then the simple injections of salt solution or boric acid may be relied upon to complete the cure.

In the severer cases rest in bed must be required and any digestive disturbance treated by proper dietetic measures. Where there is much tenesmus after the movements suppositories of cocaine, 0.015 to 0.06 gm. (1 to 1 grain), may be employed for relief. For the excoriation about the anus the oxide of zinc ointment is the best application.

#### PROLAPSE OF THE ANUS AND RECTUM.

There are three degrees of prolapse of the rectum: 1. A protrusion of the mucous membrane of the rectum through the anus, which is usually a prolapse of the anus. 2. A protrusion of the whole rectal wall through the anus. 3. An invagination of the upper part of the rectum into the lower, with protrusion of the invaginated part. This should be considered as an intussusception.



**Etiology.**—Prolapse of the rectum is seen most often in children two or three years of age. Any condition that produces frequent violent straining may cause prolapse, especially phimosis, contracted meatus urinarius, stone in the bladder, cystitis, chronic constipation, diarrhea, polyp of the rectum, violent coughing, as pertussis, etc.

It appears that there must also be some weakness of the levator ani and the anal sphincter in these cases. It is frequently found in children whose nutrition is poor and whose muscles are weak, especially in the marantic.

**Symptomatology.**—With prolapse of the rectum a tumor appears at the anal orifice. In the simpler form this is nothing more than a fold of mucous membrane surrounding the anal orifice. When the whole wall of the rectum protrudes a flattened, conical tumor is formed, the base being at the anal margin, the flattened top surrounding the central orifice. The mucous membrane covering the tumor appears dark red or purple, covered with mucus, and not infrequently showing more or less ulceration or fissures. There is regularly some bleeding from the exposed surface and a free discharge of mucus. At first the tumor appears only with defecation and is easily reduced. Later, the prolapse occurs at other times, the tumor is reduced with more difficulty, and may remain down constantly. There is little or no obstruction to defecation, but the irritation, pain, and discharge of mucus and blood weaken the patient and he becomes pale and loses flesh and strength. The sphincter ani is regularly greatly relaxed and patulous; but in some cases the prolapsed portion of the rectum may be constricted by the sphincter and sloughing ensue.

**Diagnosis.**—The diagnosis of the condition is made on examination. Polyp of the rectum and hemorrhoids have been confused with prolapse. A polyp is a single, isolated, and pedunculated mass. Hemorrhoids are not common in children and never form a complete ring. They have the characteristic appearance of dilated veins. The intussusceptum in an intussusception may appear at the anus, but the constitutional symptoms of this condition and the presence not only of a central opening, but of a space between the intussusceptum and the rectal wall, render differentiation easy.

**Treatment.**—In the milder cases relief of the cause of straining may be sufficient to cure the prolapse. If the prolapse occurs only at the time of defecation, the bowels should be kept moving easily and the child should be made to have all its movements lying on one side, the erect and sitting position always favoring the prolapse. It is sometimes useful to support the anus during defecation by pressure at the sides or by drawing the skin tightly to one side.

When the prolapse occurs constantly, it may be prevented by keeping the child in bed and strapping the buttocks tightly together or putting on a firm T-bandage.

The prolapse must be reduced whenever it occurs. Usually this is easily accomplished, but if the tumor is large it may be necessary to apply fomentations or ice for a time to reduce the swelling, or an anesthetic may be required.



If the prolapse is due to diarrhea and tenesmus, the straining may be relieved by sponging the anus with cold water or inserting suppositories of cocaine 0.015 to 0.06 gm. ( $\frac{1}{4}$  to 1 grain).

Astringent injections of tannin, 2 to 4 gm. to 30 c.c. ( $\frac{1}{2}$  to 1 drachm to the ounce), or the infusion of quassia, 60 to 120 c.c. (2 to 4 ounces), may be employed once or twice daily in the severer cases.

Holt recommends local injections of strychnine sulphate, 0.0006 gm. ( $\frac{1}{16}$  of a grain), twice daily, for a child of two years, to improve the tone of the sphincter and levator ani.

Where other measures fail cauterization may be employed. With the actual cautery four or five narrow lines are drawn from the centre of the prolapsed portion to the margin, only the mucous membrane being burnt through.

The tumor is then reduced and a pad applied to prevent recurrence. The resulting cicatrization usually cures. As a substitute for this procedure, wedge-shaped areas of the mucous membrane may be excised and the edges brought together (Ashby and Wright).

In nearly all cases success can be had by the patient application of the milder measures, most of the cases being cured in a few weeks, but from time to time obstinate cases are seen which will yield only to the operative procedures.

### POLYPUS OF THE RECTUM.

Polypi of the rectum are more common in childhood than at any other time of life. The cause of the growths is unknown. Huber has observed their association with adenoids. They are a fairly frequent cause of bleeding from the rectum in children.

**Pathology.**—Pathologically the tumors are classed as fibromata, or myxofibromata, or adenomata. The fibrous tumors are usually smooth on the surface, sometimes excoriated, and may be sessile, but are often attached by a long, thin pedicle. The adenomata are granular or warty in appearance.

The tumors vary in size from that of a pea up to that of a cherry or hazel-nut. They may be either single or multiple. The anterior wall of the rectum about an inch from the anus is the common seat of these growths, but they may occur at any part.

**Symptomatology.**—Polypi of the rectum produce irritation, with tenesmus and discharge of mucus or blood. Blood, when passed, is rarely mixed with mucus, is usually quite clear, and may amount to a drachm or more. The repeated bleeding may produce anemia. At times the polyp may be protruded through the anus and its pedicle constricted, so that the tumor sloughs off and is passed in a stool. In other instances the dragging of the tumor produces a prolapse of the rectum.

**Treatment.**—The pedunculated tumors may be simply twisted off or may be ligatured and snipped off with the scissors. The operation may require anesthetization and the use of a speculum.

Sessile polypi may give rise to no symptoms. In mild cases they may be treated by astringent injections of alum, a 1 per cent. solution, injected once or twice daily. In severe cases the base of the growth may be ligatured, and, after removal of the mass by the scissors, cauterized.

#### HEMORRHOIDS.

Hemorrhoids are decidedly uncommon in children, but both the internal and external varieties have been observed. They are nearly always dependent upon chronic constipation, and in that case are most likely to be external. The symptoms produced by piles in children are practically limited to slight hemorrhage accompanying movements of the bowels, particularly if the stools are hard and passed by straining. In rare instances the tumors are protruded.

**Treatment.**—The relief of the constipation is usually all that is required. Holt has never seen hemorrhoids in a child necessitating operative treatment.

#### ISCHIORECTAL ABSCESS.

Ischiorectal abscess is not uncommon in children. The abscess results from infection of the lymph nodes or the cellular tissue of the ischiorectal region. The source of infection is the rectum, in which there may be active inflammation or ulceration. Not infrequently ischiorectal abscess arises without its being possible to demonstrate the source of the infection. The symptoms are those of abscess anywhere, fever, local redness, swelling, pain, and tenderness. The abscess may present externally or be felt bulging into the rectum on examination with the finger.

**Treatment.**—The treatment consists in laying open the abscess, cleansing it with hydrogen peroxide, and packing it. The abscesses regularly heal promptly. Fistula in ano is rarely produced in children.

#### INTESTINAL WORMS.

These intestinal worms or parasites comprise quite a number of animals of the lower orders which exist in the mature condition in the intestine of man, drawing their nourishment either from the host's blood or from the contents of the parts in which they live. Infection is said to be more frequent among children than among adults, but except among recent immigrants intestinal parasites are uncommon in either class in this country. The parasites most frequently found in children are: 1. Cestodes, including *tenia solium* (Fig. 56); *tenia saginata*, or *mediocanellata* (Fig. 57); *bothriocephalus latus* (Figs. 58 and 59). 2. Nematodes: *ascarides*, *ascaris lumbricoides*, *oxyuris vermicularis* (Figs. 60, 61 and 62). 3. Strongyloides, *ankylostomum duodenale* (Figs. 63, 64, 65 and 66).

**Tenia or Tape-worm. Modes of Infection.**—Each of these teniæ passes through a life cycle of three stages: 1. The egg, 2. The embryo or larva, 3. The mature worm. The full-grown parasites are found only in the intestinal canal of man. Eggs are passed from any of the segments of the worm, mingle with the feces, and are passed from the host. For its further development the egg must be taken into the alimentary tract of another animal. Thus the egg of the *tenia solium* finds its refuge in the intestine of the hog. There the capsule of the egg is dissolved, the egg develops into an embryo which passes through the intestinal wall into the muscles of the host, by virtue of certain small, hook-like processes on the head, and then becomes encysted. An animal whose muscles are full of these encysted larvæ is spoken of as "measly" or "measled." The larvæ remain in this state until the

FIG. 56



1 Head of *tenia solium*; magnification, 50; 2, 3, mature and semimature segments, natural size; 4 two proglottides with uterus, twice magnified. (From Ziegler, after Leuckart.)

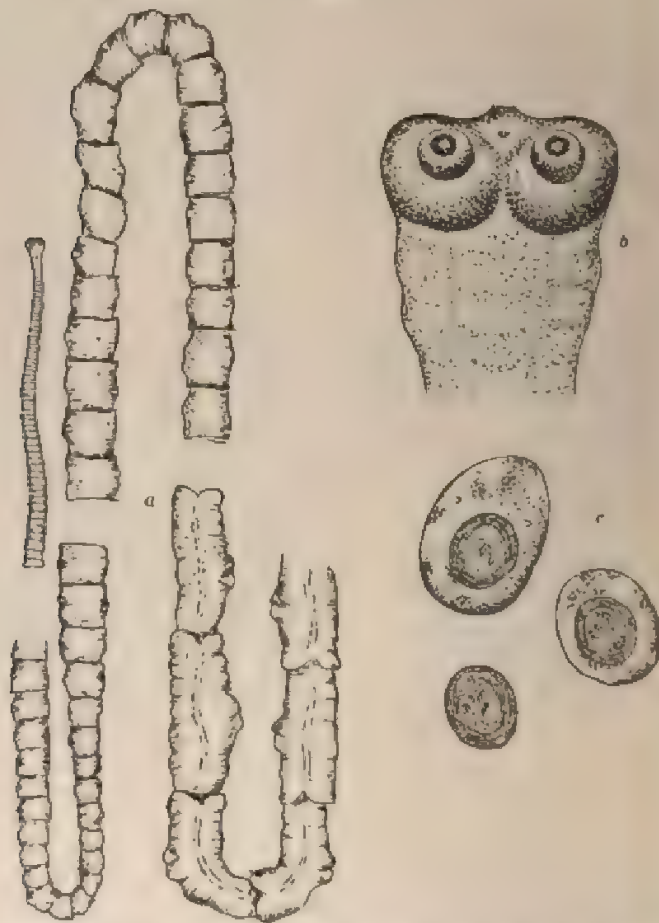
flesh of the host is consumed as meat by man, when they are set free in the intestine, where they develop into mature worms and their life cycle is complete. For the *tenia saginata* cattle are the intermediate hosts, for the *bothriocephalus latus* fish.

It is possible that man himself, taking the ova of any of these worms into the stomach, may become the intermediary host—i. e., lodge the larval form. This is known to happen in the case of *tenia solium*, the encysted larvæ of which, the *cysticercus cellulosæ*, are sometimes found in human muscle, the brain, etc.

***Tenia Saginata or Mediocanellata.***—This is the most common tape-worm in Europe and this country. Infection occurs through eating "measly" beef. The length of the parasite varies from 4 to 8 metres

(15 to 30 feet). The head is surrounded by four pigmented suckers. The individual segments are quite thick and opaque, and diminish in size toward the head, the largest measuring 2 to 3 cm. Each segment contains a many branched (20) uterus. The ova are slightly oval, or round, are yellowish brown in color, have a thick capsule, and measure 30 to 40 x 25 to 35 microns.

FIG. 57



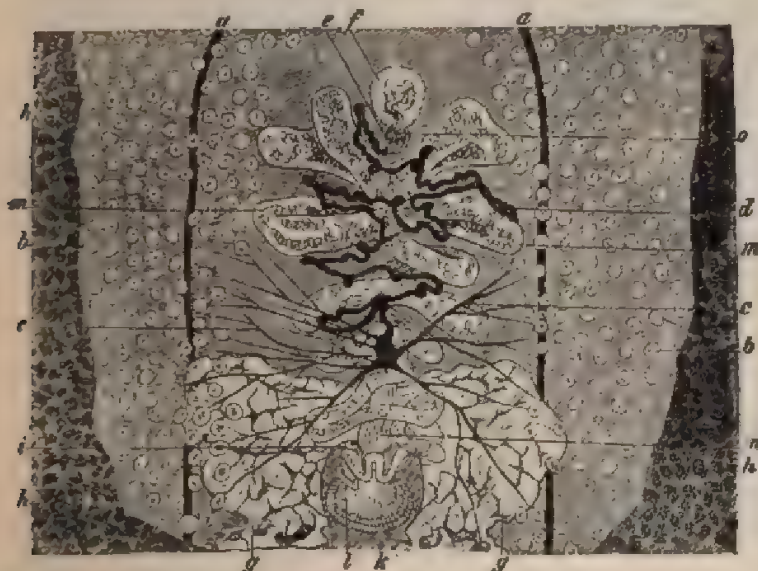
*Tachia saginata*: a, natural size; b, much enlarged; c, ova much enlarged. (Simon.)

*Tania solium* is shorter than the *tania saginata*, measuring 1 to 2 to 3 metres, as a rule, rarely reaching a length of 6 to 8 metres. The head is very small, is provided with four suckers, and with a rostellum armed with a double row of hooklets, twenty-four to twenty-six in number. The mature segments measure from 1 to 1.5 cm. in length, 6 to 7 mm. in breadth, and contain a uterus with only five to seven branches. The ova are round, of a brownish color, are surrounded with a thick, rad-



streaked membrane, and in their interior the hooklets of the embryo can usually be made out. Their diameter is 30 to 35 microns. They can hardly be distinguished from the ova of the saginata. The life cycle of the *tænia solium* is the same as that of *tænia saginata* except that the hog is the intermediary host.

FIG. 58



Middle piece of a proglottis of *bothriocephalus latus*, seen from the dorsal surface, the external layer almost completely removed: a, lateral vessels; b, seminal vesicles; c, seminal ducts; d, vas deferens; g, generative glands; h, yolk chambers lying in the cortical layers; i, collecting tubules of yolk chambers; l, commencement of uterus; m, coils of the uterus filled with eggs; n, vagina; o, vaginal opening.

In rare instances the segments of the *tænia solium* have been taken into the human stomach, either by being swallowed or by being carried back from the intestine by reverse peristalsis. In such cases the ova are set free in the stomach, the embryos develop there and thence pass into the blood stream, and so come to lodge in the tissues, muscles, brain, skin, etc. Thus man becomes the intermediary host. The encysted embryo or cysticercus, as it is called, then forms an elliptical or roundish, transparent vesicle, from 1 to 10 mm. in diameter. In its interior the characteristic hooklets may be seen.

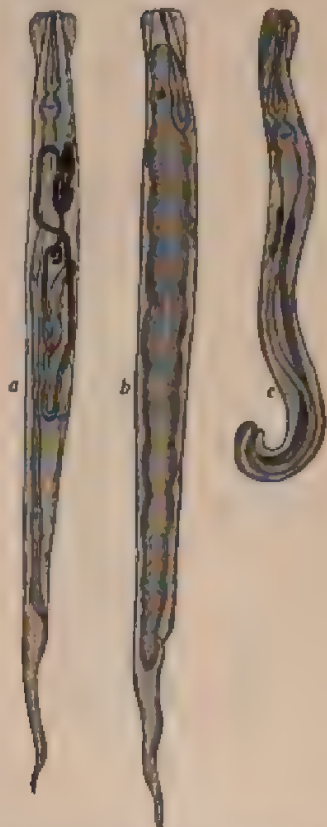
*Bothriocephalus latus* ordinarily measures from 6 to 8 metres, but may reach a length of 15 to 20 metres. Its head is shaped like a bean, and upon its flat surface are two grooves, which probably act as suckers. The uterus shows from four to six convolutions on each side. The eggs are oval, 0.07 mm. long by 0.045 mm. broad.

FIG. 59



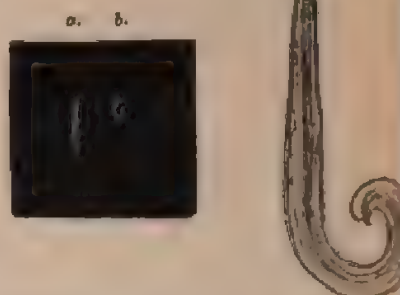
Eggs of *bothriocephalus latus*; the one to the right after discharge of yolk. (After Leuckart, from Ziegler.)

FIG. 60



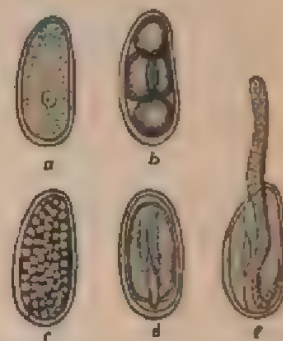
*Oxyuris vermicularis*: a, sexually mature female; b, female filled with eggs; c, male. Magnification, 10. (After Heiler, from Ziegler.)

FIG. 61



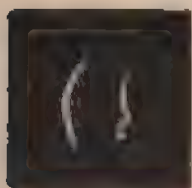
1, *Oxyuris vermicularis*: a, male; b, female; natural size. 2, Magnified.

FIG. 62



Eggs of *Oxyuris vermicularis* in various stages of development: a, b, c, division of the yolk; d, tadpole-like embryo; e, worm-shaped embryo. Magnification, 250. (After Zenker and Heiler, from Ziegler.)

FIG. 63



*Ankylostoma duodenale*, male and female. Natural size. (From Mosler.)

FIG. 64

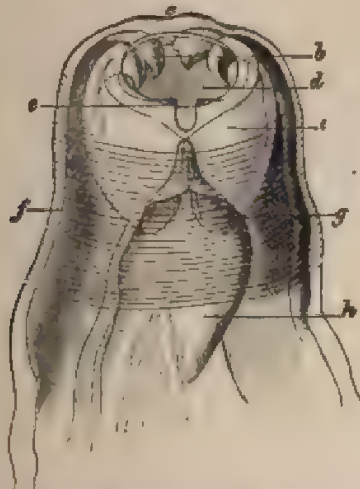


Eggs of *Ankylostoma duodenale*: a-d, various stages of segmentation; e, f, eggs containing embryos. Magnification, 300. (After Permeato and Schultze, from Ziegler.)

The larva is found in various fishes, especially the pike, perch, trout, and turbot. It is, therefore, most frequently found in lake regions. The habitat of all these tape-worms is the small intestine of the host. There is usually but one worm, but two or more have been met with.

**Symptomatology.**—There are no distinctive symptoms of the presence of a tapeworm in the intestine. The picking at the nose, restless sleep, and other symptoms popularly ascribed to worms are symptomatic only of an intestinal indigestion, and may come from overeating, improper food, etc. Often the first and only sign of the presence of the worm is the passage of some of the segments. In other cases there is indigestion, with abdominal discomfort or pain, heavy breath, and sometimes diarrhea. Nervous symptoms are seen but are in no way characteristic. The *bothriocephalus latus* sometimes produces a very severe anemia,

FIG. 66



Head of *ankylostoma duodenale*: a, buccal capsule; b, teeth of capsule; c, teeth of dorsal sucker; d, oral cavity; e, ventral prominence; f, muscle layer; g, dorsal groove; h, esophagus. After Buchanan, from Ziegler.)

at times an apparently pernicious anemia. The presence of tape-worm is regularly associated with a moderate eosinophilia.

**Diagnosis.**—This is made in nearly all instances by the segments of the worm being found in the stools. It may be made by the discovery of the eggs in the stools in suspected cases.

**Treatment.**—This is usually simple and the result satisfactory. The child is given a light supper and a dose of castor oil; then in the morning (fasting) 4 c.c. (1 drachm) of the oleoresin of male fern is given in four doses of 1 c.c. (15 minims) each (in capsule) at hourly intervals. An hour after the last dose a full dose (a tablespoonful) of castor oil is given. The worm will usually be passed promptly. Care should be taken to examine the segments passed, in the hope of finding the head. This may be difficult to do, but unless the head is found we cannot



be certain of a cure. If it remains the worm will grow anew, and after two or three months segments will be passed again. The child must be in bed during this treatment.

In children who cannot take capsules, 1.5 to 2 c.c. (20 to 30 minims) of the ethereal extract of male fern may be given, with 15 c.c. (half an ounce) each of mucilage of tragacanth and water.

If male fern is not successful kamala may be given with it in the following form:

R—Kamala . . . . .	2.0 gm.	(gr. xxx).
Syr. acacia . . . . .	3.0 c.c.	(5ij).
Misce et adde . . . . .		
Oleoresina filicis . . . . .	4 3.0 gm.	(5j ij).
Aqua cinnamomi . . . . .	30.0 c.c.	(3j).

Sig.—To be taken in two doses with an interval of three hours.

Turpentine can be given in this form:

R—Olei terebinth., . . . . .	ss	15.0 c.c.	(3ss).
Mellis . . . . .	ad	90.0 "	(Mj).
Mucilag. acacia . . . . .	q. s. ad	90.0 "	(Mj).

Sig.—8 c.c. (two teaspoonfuls) every six hours. Every second day a purge of castor oil should be given with this.

Kamala may be given in honey or molasses, 4 gm. (1 drachm), for a dose, and naphthalin in doses of 0.12 gm. (2 grains), twice a day, has been recommended.

**Ascaris Lumbricoides.**—The *ascaris lumbricoides* or round-worm is the most frequently found intestinal parasite in children. It is a cylindrical worm, looking much like the ordinary large angle-worm, except that the body is somewhat larger and the extremities more pointed. The head consists of three projections or lips, which are provided with fine suckers and teeth. The male measures about 215 mm., the female 400 mm. in length. The tail end of the male is rolled up on its ventral surface like a hook and provided with papillæ. The eggs are yellowish brown in color, almost round, and measure 0.06 mm. by 0.07 mm. in size; they are surrounded by an irregular albuminous envelope, which is covered by a tough shell; the contents are coarsely granular. There are regularly more than one of these worms present, and there may be great numbers, so that the worms may form a mass sufficient to obstruct the intestine. These worms are great wanderers. They may pass into the stomach and be vomited; they may crawl out of the nose or mouth, or pass out by way of the Eustachian tube and ear; they have produced death by passing into the larynx; they have caused jaundice by obstructing the bile-duct, and have been known to produce abscess of the liver and intestinal obstruction or appendicitis.

**Symptomatology.**—The symptoms of *ascaris* infection may be none at all, the worms or their eggs being found in the stools accidentally. In other cases there may be vague digestive disturbances, such as are described under tape-worms. These worms may, as noted above, produce symptoms by their mechanical action. Nervous disorders are not uncommon with *ascaris*, and may be severe. Among these, restlessness



irritability, sleeplessness, grinding the teeth at night, picking the nose, headache, vertigo, chorea, and even convulsions may be enumerated. In these conditions the presence of the worms in the intestine seems to act as a reflex excitant of the nervous system. Some observers believe that these nervous symptoms are produced by the action of poisons produced by the worms in the intestine.

Eosinophilia is observed in connection with the presence of ascaris in the intestine.

**Diagnosis.**—The presence of round-worms is often first recognized by the passage of one or more in the stools. In a suspected case their presence or absence can be determined by the microscopic examination of the stools for the ova. If the ascaris is present the ova can be found in large numbers. After treatment the examination should be repeated to make sure that all the worms have been expelled. The presence of an eosinophilia, not otherwise accounted for, should lead to the examination of the feces for ova.

**Treatment.**—Santonin is most effective and is most easily given. It can be combined with calomel to advantage. A child of five years may be given from 0.18 to 0.36 gm. (3 to 6 grains) combined with an equal amount of calomel. The medicine is best given in the morning on an empty stomach. It may be ordered in the following form:

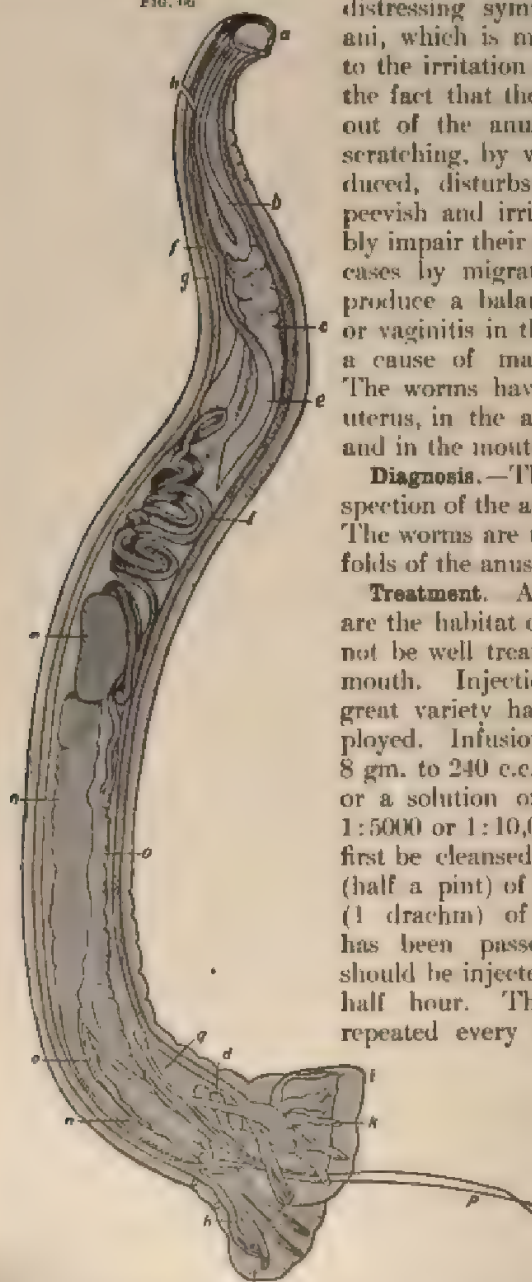
R Calomel,  
Santonin . . . . . *ad* 0.03 gm. (gr. ss).  
M. et fiat trochisci vi.  
Sig. —One tablet every half-hour until all are taken.

The child should be in bed during the day of treatment. Usually several worms are passed after such medication. If the examination of the feces shows that worms are still present, the treatment may be repeated. It should be remembered that the administration of santonin is sometimes followed by visual disturbances.

**Oxyuris Vermicularis or Thread-worm.**—These are minute, thread-like worms, the male being 4 mm., the female 10 mm. in length. The eggs are oval, 0.05 by 0.02 or 0.03 mm. in size, and covered by a membrane with a double or triple contour, the interior being coarsely granular. The female worm lives in the cecum, but after impregnation travels down to the rectum. The minute worms are present in enormous numbers in the rectum; both ova and worms are passed in the feces and are found about the anus, on the genitals, and surrounding parts. There is abundant opportunity for a child to infect its hands and so directly reinfect itself. In other cases infection may be conveyed indirectly through the agency of toys, fruit, etc. Some hold that the whole life cycle of the oxyuris may be completed in the colon, the worm finding favorable conditions in the mucous coating of the wall of the colon. Other authorities deny this and maintain that the ova must be swallowed and the embryos developed in the small intestine.

**Symptomatology.**—The symptoms produced by the oxyuris are due to the local irritation of their presence. They may excite a catarrhal colitis or proctitis, with the production of much mucus. The most

FIG. 66



Male of *ankylostoma duodenale*: a, head; b, esophagus; c, gut; d, anal glands; e, cervical glands; f, skin; g, muscular layer; h, excretory pore; i, trilobed bursa; k, ribs of bursa; l, seminal duct; m, vesicula seminalis; n, ductus ejaculatorius; o, female's groove; p, penis; q, penile sheath. Magnification, 20. (After Schultze, from Ziegler.)

distressing symptom is usually pruritus ani, which is much worse at night, due to the irritation of the rectum and also to the fact that the worms at that time pass out of the anus. The itching leads to scratching, by which ulcers may be produced, disturbs sleep, renders children peevish and irritable, and may considerably impair their general health. In other cases by migrating to the genitals they produce a balanitis in the male, vulvitis or vaginitis in the female. They may be a cause of masturbation in either sex. The worms have also been found in the uterus, in the appendix, in the stomach and in the mouth.

**Diagnosis.**—This is at once made by inspection of the anal region or of the stools. The worms are usually easily found in the folds of the anus.

**Treatment.** As the colon and rectum are the habitat of these worms, they can not be well treated by remedies given by mouth. Injections are required and a great variety have been successfully employed. Infusion of quassia, salt solution 8 gm. to 240 c.c. (2 drachms to 8 ounces) or a solution of bichloride of mercury 1:5000 or 1:10,000. The bowels should first be cleansed by an enema of 250 c.c. (half a pint) of water, containing 4 gm. (1 drachm) of borax, and after this has been passed the curative enema should be injected and retained for one half hour. These injections must be repeated every other night for a week.

This treatment may be combined with the administration of saltonin as for the ascariis for the purpose of bringing down any worm which may be lodged in the small intestine.

The treatment is usually promptly effective but from time to time cases are met with

which resist all treatment. Holt says that he has known a case which had resisted all other treatment for two years to be promptly cured by injections of a decoction of garlic and the free use of garlic by mouth. Osier mentions the case of a man who suffered from childhood until his fortieth year from these parasites.

For the itching of the anal region, which is excited by these parasites, the application of vaselin or of a mercurial ointment may be tried.

**Ankylostomum Duodenale or Uncinaria Duodenalis (Hook-worm).**—This small worm is known as one of the most dangerous parasites met with in the human being. It has long been known to occur in various parts of Europe, Egypt, and the West Indies. Within the past few years the investigations of Stiles have shown that the parasite can be found in large numbers of the children in our Southern States. Fig. 66).

The male is 6 to 12 mm. in length; the female 10 to 18 mm. The mouth capsule is hollowed out and surrounded by four sharp teeth, with which it fastens on the intestinal wall. The eggs are oval in form, 0.05 to 0.06 by 0.03 or 0.04 mm. In the interior of the egg two or three segmenting bodies are found which rapidly develop into embryos outside the body, so that after twenty-four to forty-eight hours embryos may be found in the same feces in which the eggs were observed, or fully developed ova may be found after allowing the feces to stand only a few hours (Simon). The embryos can exist for as much as thirty days outside the body. Infection may occur directly, but is probably indirect in most instances through the water, or such articles of food as cresses, lettuce, etc. The jejunum is the habitat of the parasite. It does not remain fixed in one spot, but moves from place to place. There are usually a number of the parasites present, as many as 1700 having been counted in one case.

**Symptomatology.**—The symptoms of hook-worm disease are those of a grave anemia. The children are pale and thin, the abdomen protrudes; they suffer from edema of the extremities, shortness of breath, and palpitation. Many of them are mentally dull, languid, and backward; are unfitted for school-life and unable to work. The habit of dirt-eating is common among them. They have notably capricious appetites; the bowels are constipated, and the stools show traces of blood from the hemorrhage produced by the parasites.

According to Stiles' investigations infection with this parasite constitutes a scourge in parts of the Southern States, by which many of the children are condemned to lives of illness and uselessness.

**Diagnosis.**—The diagnosis is made on the characteristic appearance of the patients and the examinations of the stools for the ova. The ova are described as twenty times the size of a red blood corpuscle, oval in shape, with a transparent, colorless, but distinct capsule, and a gray or brown, segmental protoplasm.

**Prognosis.**—This is uniformly good under appropriate treatment.

**Treatment.** Male fern may be given as for tape-worm, but thymol is more generally employed and recommended. It is to be given in

capsules, containing from 2.5 to 4 gm. (40 grains to 1 drachm) under the same conditions as any other anthelmintic. No solvent, such as oil or alcohol, is to be allowed for some time after its administration. Serious symptoms of poisoning have appeared in some cases, but the results of treatment have always been satisfactory. The anemia is to be combated with iron.



## SECTION V.

# DISEASES OF NUTRITION.

By GEORGE M. TUTTLE, M.D.

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### CHAPTER XIV.

RACHITIS—SCORBUTUS—MARASMUS.

#### RACHITIS.

RACHITIS, or Rickets, is a chronic nutritional disorder of the whole organism. Rachitis, from the Greek for "the spine," points to the mistaken idea that it is solely a disease of the bones. Modern pathology, however, teaches quite positively that, while the bone lesions may attract the most attention, the muscles, ligaments, mucous membranes, nervous system, some of the viscera and the blood show marked departures from normal. As a matter of fact, in well-marked cases of the disease probably every tissue and organ is more or less involved.

**Etiology.**—With relation to the causative factors in the disease, it is agreed that the error is a dietetic one, but beyond this we cannot advance so surely.

Rickets is rarely seen before the sixth month of life, is most common during the second year, and only its results are seen after that time. Virtually no new cases develop after a child has been well fed for some months on a more or less general diet, and in children having the disease it spontaneously disappears under these same circumstances.

Rickets develops in exclusively breast-fed babies, in babies fed on cows' milk variously prepared, and especially in babies fed on condensed milk or on the proprietary foods; so that no one form of food can be singled out as *the* cause of this disease. Originally the lack of lime-salts in the food was looked on as causal, in the days when the osseous lesions of the disease only were recognized; later, the absence of fat and again the presence of lactic-acid-forming elements in excess were considered of the greatest importance.

In the light of the best present knowledge of the physiological chemistry of digestion and nutrition, we consider the deficiency of no one of

the proximate principles in the food of so much moment as that the food should contain all the main ingredients in somewhat nearly the proportions found in normal average human milk. To be more exact, the proteids, with their chemically combined salts, the fats, and the carbohydrates should be furnished to the child month in and month out in the ratio designed by nature to supply the proper quantity and quality of food for the growing organism.

In searching for the cause in individual cases not only should we know what food the baby has been taking, and for what length of time, but we should also have an analysis made of this food. We will ordinarily find some striking defect. For instance, when rickets is seen in breast-fed babies it will usually be found that lactation has been unduly prolonged until the combined proteids and salts have become quite deficient in nutrient qualities, or the mother may be very badly nourished herself, or may be pregnant, and in either case will furnish milk showing on analysis decided departures from normal.

If cows' milk is the food, we will usually find the milk of very inferior quality, or wrongly diluted, or excessively sterilized.

In the case of condensed-milk feeding, or the use of the proprietary foods, the cause is more evident, as these foods are distinctly lacking in fats and proteids and contain excessive quantities of carbohydrates.

While the dietetic cause is all important in the development of rickets, and we see cases in which no other reason for the disease is evident, still we cannot overlook the fact that there are many contributing factors of more or less importance. One of the first to be mentioned is racial. In this country, at least, the disease is more frequent among the negroes and Italians, probably because both races as seen in our large cities are badly nourished and live under the poorest hygienic conditions. For years rickets was called "the English disease," and was considered almost a curiosity in the United States. But with the great massing of people in the large cities the disease has become very common among all the nationalities represented in our population, even the native born.

Rickets is far more common among the poor, the ill-fed, and those living in unhygienic homes than among the better-housed members of the community, showing the marked influence of fresh air, sunlight, and dry warmth as preventives. There is no reason to believe that heredity has any effect in causing rickets, nor do we now attach any importance to syphilis in the ancestry. In many cases it would appear that digestive disorders have some etiological relation to the disease, and while they are the cause of rickets in some cases, they are also frequently the result of this disease.

To sum up the above analysis of the important causes in the development of rickets, I ascribe most importance to deficiency of the fats, the proteids, and the salts in the food, and far less moment to lack of fresh air, sunlight, and warmth in the homes of these infants.

**Pathology.**—The most evident lesions are localized in the bones. These changes are seen both in bones formed from cartilage and in

those formed in periosteum. They consist essentially of excessive proliferation of cartilage cells, or of hyperplasia of the inner layer of the periosteum, combined with deficiency of the normal osseous formation which should follow in these locations.

In bone formed from cartilage, at the epiphyseal junctions there is increased vascularity of the parts, with swelling and thickening of the cartilaginous layer. The matrix of the cartilage is overcrowded with cells in irregular groups of disorderly arrangement, showing no disposition to lay the foundation for the proper histological development of the future bone. In addition no (or only abortive) attempts at calcification of these cartilage cells are seen. There is very little deposit of lime-salts and resultant ossification, and the growing bone is soft, yielding, excessively vascular, and presents many of the appearances seen in inflammation. The bones grow in diameter by the proliferation and subsequent ossification of the under layer of the periosteum. Here, again, somewhat similar changes are seen, but seldom in such marked degree. The periosteal cells are produced in excessive amount, but do not calcify normally, and there is produced a soft, spongy, ill-formed bone. Such periosteum is grossly quite hyperemic, and strips more easily than normal from the underlying bone. As the bone grows in thickness the medullary canal becomes formed by a gradual process of absorption of the inner layers of newly laid bone. This absorption process goes on excessively in rickets; so we have a bone with spongy wall and large medullary cavity, and hence it is weak and yielding.

In the flat bones of the skull, those formed in membrane, the changes correspond precisely to those described as occurring under the periosteum of the long bones—hyperplasia of the cells of the under layer of the membrane, and subsequently imperfect calcification and ossification.

On chemical analysis it is found that the bones from a case of rickets yield two-thirds organic matter, instead of the average one-third of normal bone, showing clearly the deficiency in the mineral ingredients.

The other organs than the bones present changes not of such a characteristic nature, but still showing in a general way evidences of malnutrition, and these changes are as important from a clinical as from a pathological standpoint.

The blood in uncomplicated cases resembles that of simple anemia—the red cells are of about the average number, but each cell is decidedly deficient in hemoglobin. The hemoglobin index is usually from 75 to 80 per cent. Some nucleated red cells are usually found. The leukocytes tend to be somewhat above their normal number. In cases with severe complications of a pulmonary or gastroenteric nature the marked changes of secondary anemia are found—great reduction of the erythrocytes combined with a low percentage of hemoglobin. The red cells are also found to undergo the various morphological changes characteristic of this condition. More or less leukocytosis is also present, due to excess of lymphocytes and a slight eosinophilia.

The heart and the voluntary muscles are all imperfectly nourished and anemic, sharing in the general weakness of all the body tissues. The ligaments become relaxed and weakened, although no structural change can be found in them.

The mucous membranes, both of the respiratory and alimentary tract, are very apt to show catarrhal inflammations, which are considered secondary to the rachitic diathesis. Whether there is any real pathological change in the structure of the mucous membranes which is responsible for the marked tendency to catarrhal complications in rickets has not yet been discovered.

The lungs in advanced cases show indentations made by the malformed or collapsed chest wall, due to a mechanical result of rickets.

The liver lies low owing to the diminished chest capacity, and is often actually somewhat enlarged. This is due probably to a passive hyperemia, which in prolonged cases may be followed by a development of new connective-tissue cells with some hardening of the organ. The spleen is regularly enlarged and hyperemic, and may undergo the same changes as the liver. Along with this we usually find the lymph nodes of the body enlarged and hyperemic. It seems that they are specially liable to infections from any slight sources of irritation.

**Symptomatology.**—It must be remembered that rickets is a chronic malnutrition, slow in onset, slow in development, and slow in recovery. Its first beginnings are often unrecognized, but this is due as much to neglect to look for the early signs as to their comparative insignificance.

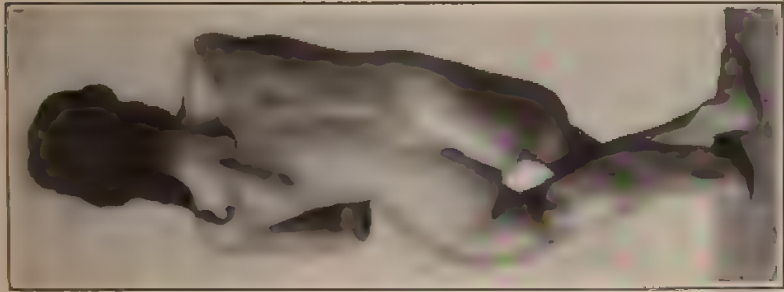
One of the first signs that should attract attention is more or less anemia in an otherwise seemingly well-nourished infant. Along with this, careful examination will show more or less feebleness of the musculature. Such a baby will make no effort to stand on what appear to be well-developed legs, the head will not be held upright, the back will be bent more than a normal baby's in sitting, or no efforts to sit up will be made. The whole muscular system will be found flabby as compared with that of a normal child of the same age. With this there will usually be a history of constipation, due probably in many cases to weak muscular action in the intestinal walls; in other cases it is the result of the character of the food.

It will also be found that the skin is soft and easily irritated and the baby sweats a great deal about its head and neck, particularly during sleep; that it rolls its head restlessly about on the pillow, with the result of almost complete baldness in the occipital region. Its sleep is restless and broken, and there is more or less general "nervousness" present, all being probably due to malnutrition of the brain.

As the disease advances the above signs become intensified, and in the large proportion of cases there are added the more characteristic changes in the bones. The first that can ordinarily be found is a slight beading of the ribs, evident only on palpation. As this progresses it takes the form of the so-called "rachitic rosary," which is easily evident to the eye. The "beads" or protuberances are due to the pathological



PLATE VI.



Rachitic Curvatures of Lower Extremities. (Solley.)



hyperplasia, characteristic of the disease, taking place at the costochondral junctions on either side of the sternum. They are found at the ends of the ribs, and the row of "beads" runs downward and outward along the costal margin. This beading is also present on the under or visceral side of the thorax, but naturally can only be appreciated here postmortem.

About the same time on careful examination similar changes can be found at the epiphyseal junctions of some of the long bones, more particularly at the wrists, ankles, and knees. There is a knob-like enlargement, palpable in the early stages, visible later, which lies exactly at the point where epiphysis and diaphysis join, and which gradually flattens down to the level of the bone on either side of it. Neither the "beads" nor the "knobs" are tender to pressure.

The head appears large and square. The forehead is high and broad and the top of the head is more or less flattened. There is a tendency to a shallow furrow along the line of the coronal and sagittal sutures. These appearances are due to the development of "bosses" on the frontal and parietal eminences. These "bosses" are the thickened growths of bone characteristic of rachitic pathological changes in bones developed in membrane. The sutures and fontanels are large and regularly late in closing, the anterior fontanel being often open at the end of the second or even the third year. The veins of the scalp are large and prominently blue in contrast to the white skin.

*Dentition* is almost always delayed, the first teeth frequently not appearing until after the first year. Then they are often cut irregularly, "crossed teething," and frequently decay early. The various disturbances ascribed to dentition are much more common in rachitic children than in normal ones.

As a result of these defectively nourished and yielding bones, various mechanical changes in shape and form follow, some the result of atmospheric pressure, others of muscular action, and others due to their inability to sustain the superincumbent weight. In the *thorax* atmospheric pressure produces a marked depression of the ribs just at the costochondral junction and parallel to the sternum. A second transverse groove is also found running horizontally around the lower part of the chest. Atmospheric pressure plus diaphragmatic pull is probably responsible for this. The sternum itself may be protruded, or may be depressed, producing the conditions known as pigeon-breast.

The vertebrae are not as hard as normal, the ligaments are relaxed, and the muscular support is deficient in tone, resulting in a bending of the spine. The kyphosis or scoliosis, so produced, forms a long, uniform curve, with none of the sharp angles seen in tuberculous disease (Fig. 67). These rachitic curvatures can usually be made to disappear by gentle traction or change in position during the disease proper. This is not true, however, of the resultant bendings that may remain as permanent deformities after the rachitis itself is past.

In the posterior or lateral regions of the head, more often over the occipital bone, there are sometimes found softened spots of imperfect

bone development called *craniotabes*. On pressure with the tip of the finger these areas dent in, but spring out again when the pressure is released. *Craniotabes* gives to the finger a feeling of crackling.

Secondary changes in the long bones regularly develop, especially in the legs. These may result in bowing outward of the tibia and femur with the production of the condition known as bow-legs, or *genu varum*, or in the opposite condition of knock-knees, or *genu valgum*. The former seems due more particularly to mechanical bending of the bone

FIG. 67



Rachitic kyphosis. (Whitman.)

while the latter consists mostly of an hypertrophic growth and consequent lengthening of the inner condyle of the femur, causing the tibia to form an obtuse angle with the femur. As a result of this, when the child's thighs are placed parallel with the knees together, the ankles are separated more or less according to the amount of the knock-knee present.

When the bones of the upper extremity become bent, the hand usually bows outward, and the radius and ulna backward. In some cases the bones are so softened and yielding that irregular and distressing deformities may develop in any of the long bones (Figs. 68 and 69).



In the pelvis rachitic changes frequently are found, but they always escape notice and are unimportant except in women at the time of labor. The commonest form of pelvic deformity of rachitic origin is a shortening of the anteroposterior diameter, due to a pushing forward of the body of the sacrum.

The ligaments about the joints are more or less relaxed and weakened, which, in combination with the poorly developed muscles, aids in the deformities and prevents such children from supporting themselves and walking as early as normal.

FIG. 68



Extreme deformities, the result of infantile rachitis. The left leg forms practically a right angle with the thigh. (See Fig. 69.) (Whitman.)

Rachitic children frequently appear fat and plump, but may be thin and badly nourished. The abdomen is enlarged and tympanitic, for which there are probably two reasons: the diminished thoracic cavity presses down the diaphragm and crowds the abdominal viscera, and the stomach and intestines are more or less distended as the result of a complicating chronic indigestion and weakened muscular walls. There is regularly no change in the heart, except that due to anemia and malnutrition, nor in the temperature. The urine, however, may present an

excess of phosphates and show traces of albumin. A bruit is often to be heard over the anterior fontanel, but this is of no special significance.

One of the marked characteristics of rachitic children is their tendency to catarrhal inflammations of the gastroenteric and respiratory tracts, and to reflex explosions of their nervous systems. So we frequently see gastritis, gastroenteritis, chronic indigestions of gastric or enteric origin; laryngitis, bronchitis, or bronchopneumonia; and laryngismus stridulus, tetany, or general convulsions developing in the rachitic.

FIG. 69



Skiagram of Fig. 68, showing the deformity to be due to distortions of the diaphyses of the bones while the epiphyses are practically normal. (Whitman.)

Furthermore, children with rickets are much less resistant to infection by the various contagious diseases than normal, and if they do develop them their mortality rate is higher than usual.

Rickets runs a course of one to two years, and most of the symptoms disappear spontaneously. The bone changes, however, are more persistent.

**Diagnosis.**—In the early stages of the disease and in mild cases it is only necessary to have rickets in mind, so as not to overlook it. The presence of anemia, muscular weakness, constipation, delayed dentition,

orsweating of the head should always call one's attention to the possibility of the beginning of rickets. With this idea in mind, a careful examination of the bony framework will usually show enough to corroborate the diagnosis.

Well-marked cases should offer no difficulty in diagnosis, except that at times it is difficult to distinguish the cranial changes of rachitis from those of moderate hydrocephalus. In the latter condition the forehead is much more prominent and overhanging, and the breadth of the whole cranium is markedly increased. The rachitic enlargement is mostly due to the thickening of the bones at the parietal and frontal bosses. The presence of other rickety changes in the body will assist in the diagnosis, as well as the backward cerebral development present in hydrocephalus. The two diseases may coexist.

The various lesions of congenital syphilis appear much earlier than those of rickets, and the later bony changes are not so regularly confined to the epiphyseal junctions as in rachitis.

In chondrodystrophy fetalis, achondroplasia, there is marked shortening of the long bones without thickening of the epiphyseal cartilages.

The pseudoparalysis of rickets is easily distinguished from real cerebral or spinal paralysis by the absence of any changes in electric reaction, or in the superficial reflexes.

Scurvy is distinguished by the hemorrhagic gums, the painful swelling in the shafts of the bones, and the prompt benefit following antiscorbutic diet. At times the diseases coexist, but the relief of the scurvy will not lessen the rachitic evidences.

**Prognosis.**—Rachitis in itself is seldom if ever a fatal disease. More than this, it is self-limited and regularly recovers of itself as the patient changes by degrees from the limited diet of infancy to the more general food list of childhood. The osseous deformities, on the other hand, which have resulted from the malnutrition of the rachitic state remain permanently to bear their witness to the infantile disease, and at times, as in the deformed pelvis of a pregnant woman, to be a source of danger to a mother and an unborn child.

But while rachitis in itself can be considered as having a favorable prognosis, it is indirectly a source of high mortality in infancy and early childhood from its complications. Rickety infants are specially prone to catarrhal inflammations of the respiratory and gastrointestinal tracts, and in such cases frequently die when a healthy child would recover. This is particularly true of bronchitis, bronchopneumonia, or whooping-cough, and also of gastroenteritis, or enterocolitis. They also frequently die in an attack of general convulsions, their badly nourished nervous systems seemingly not being able to withstand the shock. So we must not make light of any manifestation of rachitis when it is present.

**Treatment.**—Prophylaxis is of great import in this condition, and especially so when by careful observation we become convinced that the first evidences of rickets are making their appearance. Either before such appearance, or when the first suggestion of symptoms begins, every precaution should be taken with the general hygiene and the food.

The infant should live and sleep in well-ventilated and sunshiny room should be accustomed to daily outings in the fresh air, should have regular bathings in cool water, with thorough rubbings afterward, and should be warmly clothed.

More important than these is a careful regulation of the feeding. If breast-fed, the mother's milk should be analyzed and efforts made to remedy any deficiencies present in it. If lactation has been rather prolonged, or if the milk cannot be improved through hygienic measures directed to the mother, supplementary feedings of a suitable food for the child's age should be given in addition to the breast milk, and these should be increased in number even to the complete weaning from the breast in case of need. If the baby is artificially fed, a careful investigation is necessary as to the kind of food given, its method of preparation, the way it is given the baby, and its results from a digestive standpoint. Any imperfections in one or more of these particulars should be at once regulated, and the results carefully watched. In short, all of the well-known principles of the modern scientific feeding of infants and children should be systematically followed in these cases.

If rachitis has already positively developed, all the general hygienic and dietetic measures should be scrupulously carried out in the minutest detail. No care can be too painstaking in attempting promptly to put a stop to the symptoms of the rachitic malnutrition. Fresh air in the home and sleeping-rooms, abundant out-door exercise, cool bathing and massage to stimulate the respiration and circulation and to accustom the skin to changes of temperature, and so prevent the tendency these children have to "catch cold," are all of great importance.

The diet should be made to conform as nearly as possible to the suitable for a baby of the patient's age. Mother's milk may need to be supplemented by one or more daily feedings of properly modified cows' milk. Artificially fed babies will usually be found to be getting condensed milk, or one of the proprietary foods, or thoroughly sterilized cows' milk. Any of these foods should be stopped and raw, modified cows' milk substituted. Some babies will be on too diluted cows' milk not offering enough solids for proper nutrition, and others still on too concentrated cows' milk which cannot be thoroughly digested, starts up more or less gastroenteric indigestion, and is never assimilated. Others are too early fed on "table food" to the exclusion of milk and before their immature digestive organs can extract the proper nutriment from it. It is possible in virtually every case to find some gross error in feeding which calls for instant correction.

The general principles are to give the proteids and fats up to their maximum limit of digestion and absorption and to keep the carbonic hydrates a little below normal, so that there shall be a more perfect metabolism of the former two proximate principles, as they are the great tissue builders. The regular feedings can be nicely supplemented by the daily use of beef-juice, or scraped beef or mutton, for the proteids and by cream or butter for the fats. Any of these substances fulfil the



indications of easy digestibility, and supply an abundance of proteids and fats very satisfactorily.

Much more than half the battle is fought by hygiene and diet, but drugs are more or less helpful and certainly should be used except under special contraindications. The most useful drug, and the one which is most commonly prescribed is cod-liver oil. But if we analyze its rationale, we at once conclude that even in this case we are giving a *food* rather than a drug. Cod-liver oil is primarily an easily absorbed fat, and so is especially useful in rachitis. It undoubtedly does good, but it must be given with judgment and with particular attention to the digestion. A minimum dose rather than a maximum should be our aim and on the least evidence of any gastric or intestinal upset it should be stopped at once, and when resumed the dose should be smaller than before. 0.63 c.c. to 1.5 c.c. (5 to 20 drops) three times daily should represent the dose for a year-old child. Olive oil may at times be used as a substitute.

Phosphorus has been, and is, prescribed extensively in the treatment of rickets from its well-known effect in stimulating the growth and ossification of bone. Its use has the sanction of many well-known men both at home and abroad. If given judiciously it may hurry the process of recovery. It should be prescribed in doses of 0.00032 gm. to 0.00065 gm. ( $\frac{1}{16}$  to  $\frac{1}{8}$  grain) three times a day. Thompson's solution, containing 0.0032 gm. to 4.0 c.c. ( $\frac{1}{8}$  grain to the drachm), freshly prepared, seems to me to be the most satisfactory preparation to use.

Lime in some form is theoretically given to furnish more lime-salts for encouraging the calcification of the bones. Calcium hypophosphite may be given, or the precipitated phosphate of calcium, either of them in doses of 0.32 gm. (5 grains) three times a day mixed with the food. Limes-water is of no direct value in this disease.

The anemia should be treated by some iron preparation, as the *vinum ferri amarum*, 2.0 c.c. to 4.0 c.c. ( $\frac{1}{2}$  drachm to 1 drachm) three times a day, although the fresh beef-juice, not beef-tea, may be all that is necessary in combating this symptom. In the use of any or all of these drugs care must be taken not to upset the appetite or the digestion by them, and it must always be remembered that a good digestion with proper diet and hygiene is much more satisfactory in the care of rickets than any other therapeutic measure.

Complicating conditions must be met, as they arise, in the usual way. Especial attention must be paid to any digestive troubles, for the double reason of their possible danger and their harmful effect on the rachitic process. The osseous system needs attention during the activity of the rachitis to prevent, if possible, the formation of bony deformities. Much can be done by care in keeping the child in proper positions both when sitting and standing to prevent permanent kyphosis or scoliosis. Knock-knees and bow-legs can be more or less prevented by not urging too early attempts at creeping or walking, and the possibility of helping to cause bow-legs by too bulky diapers should always be kept in mind.

Often the use of light supports or braces may be of distinct advantage, but should be supplemented by massage and passive exercise.

In the treatment of marked deformities due to a pre-existent rachitis the case should be considered one for the use of extreme orthopedic or surgical measures. The results of rickets in the pelvis belong to the domain of the obstetrician.

### SCORBUTUS.

This disease, of recent years recognized rather frequently among infants, is the old-fashioned sea-scurvy, produced by the conditions of modern life which lead to the necessity for the frequent artificial feeding of infants. Scurvy is a constitutional malnutrition characterized mainly by anemia and a general hemorrhagic tendency, and definitely connected with a rather prolonged period of improper feeding. While it has only been recognized properly for about twenty years, as occurring in infants, before that time many cases were reported as acute rickets, or as hemorrhagic periostitis, or under other names, which were undoubtedly infantile scorbutus. Its association with rachitis in the same child led for years to much confusion in diagnosis, and hence to imperfect classification.

**Etiology.**—Infantile scurvy develops with greatest frequency from the fourth to the twenty-fourth month of life. An occasional case is seen before the fourth month, but rarely, as the conditions leading to its development require some little time to produce the scorbutic symptoms. After the second year cases are also reported, but with much less frequency, and, of course, they cannot be called "infantile" after that time.

It is in the large proportion of cases a disease of the middle and upper classes, thus contrasting with rachitis, which is distinctively a disease of poverty. This is probably explained by the comparatively small number of babies among the poor who are exclusively bottle fed, and the early age at which these same babies begin to eat solid food of one kind or other, particularly potato, which is recognized as one of the best antiscorbutics. On the other hand, infants in the middle and upper walks of life are much more frequently bottle-fed entirely, and often on the very foods which are most prone to cause scurvy.

Previous health seems to have very little bearing on the disease, nor does the presence or absence of good hygienic surroundings influence it. As many of the cases develop among the well-to-do, naturally the hygienic environment is usually above the average.

In studying the etiological factors diet must be considered first and foremost, and as a matter of fact this is the only actual causative agent. But the special form of diet used previously to the development of symptoms is so varied and so beyond classification, that it is difficult to draw definite conclusions which will convict any one food as containing some positively harmful element, or as lacking some principle

necessary to nutrition. Even what is ordinarily considered perfect infants' food, mothers' milk, has some few cases of scurvy charged up against it. Raw cows' milk must be put in the same category. Sterilized, pasteurized, or peptonized milk has, on the contrary, each many cases to its credit. A few cases are reported as developing in infants fed on "table food," although the kinds of food and their method of preparation are unknown, and were probably entirely unsuitable for babies. The vast majority of cases, however, give the history of having been fed on some one of the proprietary foods, or on condensed milk, and usually in numbers proportional to the frequency with which the individual form of food is used. This looks not as if any one of these foods was at fault, but as if the whole class of "preserved" or "dead" foods lack something necessary to prevent scorbutus. The lack of the quality which is best called *freshness* seems to be the most common fault in the large number of these foods which are responsible for the great majority of the cases. Virtually the only cases in which this is not lacking are the few with the history of feeding at the breast or on raw cows' milk. For these it is difficult to make any explanation except that the food was low in proteids.

In a general way we can say that the antiscorbutic agent is something vital and something that seems to be destroyed by drying, by preserving, or by excessive heating. Probably some future investigations into the biochemistry of foods will unravel this vexed point, determine whether there is a primary intestinal toxemia, and probably find the exact element necessary to prevent the development of scorbutus.

For the present we must adopt the conclusions of the American Pediatric Society's collective investigation that "scurvy follows the prolonged employment of some diet unsuitable to the individual child," "that there are certain forms of diet prone to be followed by scurvy," and "that the farther a food is removed from the natural food of a child the more likely is its use to be followed by the development of scurvy."

**Pathology.**—As the results of treatment are so strikingly successful, fatalities are rare and postmortem examinations more so. The changes that are found are almost all those due to hemorrhage in some form or other. These are most marked under the periosteum of the long bones. A hematoma forms there and strips the periosteum from the bone, and in advanced cases causes a separation of epiphysis from diaphysis. For some reason these subperiosteal hemorrhages seem more common about the shaft and lower end of the femur than elsewhere, but similar changes occur over the tibia and other long bones as well as on the scapula and along the anterior margin of the ribs. The bone in the neighborhood of the epiphysis is regularly congested and hemorrhagic.

Hemorrhagic spots are also seen in the pleura, pericardium, on the liver, or other viscera, and subcutaneously in almost any part of the body. The gums are swollen, edematous, and hemorrhagic, and the teeth are frequently loosened.



**Symptomatology.**—Ordinarily the infant presents evidences of anemia and malnutrition which may be marked enough to attract the attention of an unskilled observer, but in some cases these signs can be discovered only by an expert. As the same general improper diet that is responsible for scurvy is also causative of rickets, we may find evidence of the latter form of malnutrition present, although I believe that there is no definite connection between the diseases, and scurvy is seen in children showing not the least sign of rickets. In the past the two diseases have been frequently confused, and even now by some are considered cognate, although the only real reason for doing so is that the same diet may cause either or both diseases. We must remember, too, that scurvy is essentially a chronic disease in its inception, and so the afflicted infant should be expected to show more or less malnourishment before the characteristic symptoms appear.

The symptoms show themselves first usually by pain and tenderness. The pain may be spontaneous and present even when the child is at rest, or may only be evoked by motion or handling. The little patient often screams when anyone approaches as if in dread of the possible pain, or when any motion of the bed is made that shakes him. On examination it will usually be found that this tenderness is limited to the limbs, more commonly the lower, and that motion of other part of the body can be made without eliciting the symptom. Occasionally the spine is sensitive and tender.

As a direct result of this tenderness there is found a false or voluntary paralysis of the limbs. The child unconsciously holds them quiet to prevent suffering. This so-called paralysis is ordinarily not flaccid but spastic, the muscles being contracted usually in flexion and in the position of greatest comfort, and it can be easily proved that no true paralysis is present.

The same limbs may also show what is really the most characteristic sign of the disease—marked fusiform swellings. These are due to the subperiosteal hemorrhages, which may be small or large, and single or multiple. They are ordinarily in the epiphyseal regions, more often of the femoral, at the juncture of the diaphysis and epiphysis, and consequently near the joints, but may be located on the shafts of the long bones. The skin is smooth and waxy, very seldom bluish or reddened. In severe cases there may be a separation of the epiphysis and hemorrhage into the joint with the signs of fracture and effusion.

The other typical symptom of scurvy is equally frequent and, perhaps, even more common. When present with the above it is almost pathognomonic. This is the swelling of the gums. In mild cases they are only swollen and brighter in color than usual with a dark reddish line close to the teeth, but in more advanced stages they become spongy, protuberant, ulcerated, and hemorrhagic. When teeth are present the gums are more seriously involved, but many cases of scorbutic gingivitis have been reported before dentition has begun, proving that teeth are in no wise necessary to this symptom. The hemorrhage may be petechial in the cases where the teeth are not erupted.



**H**emorrhagic conditions elsewhere are also frequently to be found. Ecthymoses of the subcutaneous tissues anywhere on the body are frequent, the common location being in the loose areolar tissue around about the orbit. This produces a "black eye," or a protrusion of the eyeball from its socket. Echinotic areas on the thighs and legs are also seen as well as petechiæ. Hemorrhages from one or other of the mucous membranes may be present, as from the mouth, stomach, intestines, or nose. Blood or albumin without erythrocytes is often found in the urine.

There is no regular fever associated with this disease, but irregular rises of temperature may, and often do, occur, even in the absence of complications.

The blood shows nothing characteristic beyond the regular changes always present in secondary anemia. Leukocytosis is not found except as resulting from some complicating condition. All grades of severity of the disease are encountered from simple anemia with swollen gums and tender limbs, to those with marked degrees of hemorrhagic gingivitis, large subperiosteal hemorrhages, and separation of the epiphyses.

**Diagnosis.**—Acute articular rheumatism and paralysis are often diagnosed when scurvy is the condition present. A careful examination of the gums and of the location of the seemingly swollen joints will usually prevent an error. The swelling of scurvy is seldom in the joint proper, but on the shaft of the bone at the junction of the diaphysis and the epiphysis, and the so-called paralysis can easily be proven not real, but an immobility due to pain. Scurvy has been mistaken for osteosarcoma, but the accompanying symptoms should prevent such a mistake, and the therapeutic test of treatment can always be called on as an aid, and should be tried before a serious operation is undertaken. Inherited syphilis occasionally causes a separation of an epiphysis or a pseudoparalysis of one limb. This limitation is not seen in scurvy.

Recently a case of scurvy with subperiosteal hemorrhage has been reported as operated on under the diagnosis of osteomyelitis. Such error would be unlikely if the possibility be kept in mind, and an examination for other signs of scurvy, together with dietetic treatment, would at once settle the question. The hemorrhages from the intestine should not give trouble in diagnosis if the all-important dietetic factor of scorbutus is considered. The same may be said of blood in the urine.

Lead poisoning gives symptoms of swollen gums; in scurvy, however, the blue line found in lead poisoning is absent.

**Prognosis.**—This is very good if the disease is promptly recognized and properly treated. But it must be remembered that this disease, unlike rickets, is not self-curative, but is progressive and chronic in its course, and so tends to become worse as time advances.

Unrecognized cases may get into such a condition of malnutrition and exhaustion as to be beyond hope, and so may die even with proper treatment applied late. If scurvy attacks a child already weakened by some previous disease it must be looked on as influencing the prognosis unfavorably. Pneumonia is likely to cause death in untreated cases.

**Treatment.**—Prophylaxis is of the first importance. A knowledge of the etiology of infantile scurvy and careful attention to all the details of the correct feeding of infants should absolutely prevent the development of any case of the disease. If science has not yet taught us the precise nature of the etiological factor in scorbutus, she has taught us how easily to prevent the development of the disease. All infants who are fed artificially should have some fresh unsterilized cows' milk and orange-juice three or four times a week.

If the disease has begun, the curative treatment becomes necessary. This is entirely dietetic in nature. No case of scurvy has ever been reported as cured by drugs only. Since so many different ways of feeding have at one time or another been responsible for cases of scurvy, the first rule to be noted is that a change of diet must be made. At least very palpable errors must be corrected, and they are usually so plain that it requires very little exact knowledge of infant feeding to make them. Proprietary foods, condensed milk, sterilized milk, and all "dead" foods must be at once discontinued. Raw cows' milk, properly modified for the individual baby, is ordinarily the most proper food to substitute. This in itself will usually effect a cure, as it contains the antiscorbutic property in moderate amount. But other substances contain it more generously, among the best of these being orange-juice, expressed beef-juice, and potato. The first two can be easily given to babies of any age, the potato being more suited to the treatment of older children. But it even can be given to quite young infants if it is thought advisable. The juice of a whole orange can be given daily to a baby, and the expressed beef-juice is given in doses of a tablespoonful twice daily. Potatoes are prepared by thorough steaming and then mashing through a sieve. They can be given dry or rubbed up with milk, from a teaspoonful to a tablespoonful two or three times daily, according to the age of the child. In infants a little of this may be put in the bottle. Under such dietetic regimen as this the symptoms may be confidently expected to improve greatly in a few days, and complete cure may be looked for in three or four weeks.

The child should be kept as quiet as possible to protect it from pain. The swollen limbs should be wrapped in cotton, and kept on a splint if very tender, and especially if the epiphyses have separated. The mouth should be washed clean frequently by some mild antiseptic solution to prevent bacterial decomposition in the secretions of the inflamed gums.

After improvement commences, every attention should be paid to building up the baby's nutrition by fresh air, proper bathing, massage, iron, and cod-liver oil if necessary. No disease presents itself where dietetic treatment is more satisfactory if properly carried out, and where the results of our therapeutic efforts can be used as an aid to diagnosis with such confidence.

**MARASMUS.**

**Marasmus, Infantile Atrophy**, also called *Athrepsia*, occurs very frequently among infants. Its essential feature is wasting, and this without recognizable or gross pathological lesion.

Various organic diseases of infancy produce the same resultant atrophy, and with our growing improvements in diagnosis we are often able *autemortem* to find such a cause; but even with these exclusions simple atrophy is a very frequent disease, and, more than that, a very frequent cause of infantile death. Marasmus could be best defined as emaciation occurring in an infant without discoverable cause.

**Etiology.**—Undoubtedly more than one element is concerned in the causation of this disease. In many cases the use of food decidedly unsuited to the child in quality, quantity, or method of preparation and feeding explains the development of marasmus. In other cases markedly unhygienic surroundings are the cause, and more often yet it is a combination of the two. Frequently it is easy to prove the presence of a decidedly non-resistant inherited constitution, and in any case it is almost impossible to say that this vice of development is not present.

Further, and more to the point, marasmus may be met with where neither of the first two causes is at work, and where the child is well fed on a proper diet, and lives under suitable hygienic conditions. It is in these cases that the etiological difficulties present themselves, and we are compelled to seek for some cause of a more subtle nature at work in an infant whose resisting powers are below par.

Marasmus, or infantile atrophy, is a disease of the first year of life, or, at most, of the early part of the second year. It is rare among breast-fed infants, except where the mother is so overworked and underfed as to furnish milk of most inferior quality, but is commonest in artificially reared babies, and especially in those in institutions. Indeed, infantile atrophy might well be classified as an institutional disease. In private practice, especially among the better classes, it is almost unknown. A long-continued disturbance of digestion bears a causative relationship to many cases.

No investigations have been able to associate any specific micro-organism with the etiology of this disease, but a variety of different bacteria are found in the intestinal contents, as in most infants, well or ill.

The disease is in some way a gastroenteric infection or intoxication, and that it has the power of being conveyed from one individual to another, as is seen so regularly in institutions where infants are kept together. In what way this takes place, whether through the air, or by overcrowding, or by the diapers, or by the handling of the nurses cannot be decided, but precautions should be taken to prevent each of these possible means of spreading the infection; in other words, the prevention of the poison of "hospitalism."

**Pathology.**—The disease seems to be essentially an error in the assimilative functions of the digestive tract, and, as in all functional dis-



orders, the lesions are few and seemingly unimportant. Certain observers report a sclerosis of the intestinal mucous membrane with atrophy of the glandular substance. There is hyperplasia of the epithelial covering, with connective-tissue infiltration of the substance of the mucous membrane. In areas the villi and glandular layers have disappeared. The mucosa itself is in places thinner than normal. Solitary and agminated follicles are usually enlarged and may be pigmented, giving the so-called "shaven-beard" appearance to the naked eye. The mesenteric lymph nodes are regularly enlarged, but no more so than in children dying of any gastroenteric disease. In some cases none of these microscopic nor macroscopic lesions are found, showing that they are in no wise typical of marasmus.

The results of the marasmus are more regularly found. The body is emaciated, and almost free from subcutaneous fat, causing the skin to lie loosely and wrinkled on the muscles. Petechial spots and late subcutaneous hemorrhages are quite common. The liver shows fatty degeneration, and appears enlarged in contrast to the wasted body. The kidneys frequently are the seat of parenchymatous degeneration. There is quite regularly more or less hypostatic pneumonia, especially along the posterior borders of the lungs, and with this are frequent areas of atelectasis. The heart is atrophied and pale. The stomach is of a good deal dilated and its lining membrane pale.

While these lesions represent our imperfect knowledge of the pathological anatomy of marasmus, its functional pathology is probably more important, but in many ways equally vague. It is supposed that the disorder is due to deficient digestion and absorption of the proteins and somewhat so of the fats. This vice of assimilation is supposed to result from the lesions of the mucous membranes already described.

**Symptomatology.**—The disease begins almost imperceptibly, and can only be detected at first by means of the scales at the weekly weighings. It progresses in the same gradual way as it began, but with seemingly irresistible momentum. Steady, persistent loss of weight and resulting emaciation are the most characteristic features of marasmus from beginning to end. And especially is this failure of nutrition typical when, try as we may, no evident cause for it is to be found.

The infant loses its previous plump appearance; the muscles grow soft and flabby; the subcutaneous fat disappears, leaving the skin wrinkled, dry, and hanging in loose folds on the trunk and extremities. Over the abdomen the skin can often be picked up and drawn away from the underlying fascia in much the same way as is done by the "elastic skin" men of the dime museum. The face grows thin, pinched and pale, and takes on the characteristics of senility, making these babies look decidedly like little old men. The anterior fontanel is sunken and depressed, and shows a seemingly exaggerated pulsation. While every other portion of the body wastes until it seems to consist only of bony framework covered with skin, the abdomen grows more prominent and distended, due partly to the enlarged liver, but mainly to the accumulation of gas inside the intestinal canal.



Anemia is marked, but has only the characteristics of ordinary secondary anemia with a decided fall in both hemoglobin and red cells. The pulse is rapid and feeble, and the breathing shallow and insufficient. The temperature is regularly subnormal even in the rectum. Rises of temperature occur from time to time, but are usually due to some temporary intercurrent trouble.

The tongue is coated and dry; the mucous membrane of the mouth is red and angry looking, and often shows the presence of the thrush fungus. The appetite is regularly enormous, being the expression of the demand of the starved tissues for nourishment, which no amount of food taken into the stomach seems able to appease. This is only natural, as filling the stomach in this disease does not mean feeding the tissues. This unnatural appetite leads to gastric dilatation and rather frequently to attacks of vomiting, the stomach being kept at work too continuously for its weakened state.

The bowels may be constipated or may be loose. Alternating constipation and diarrhea are fairly common. The stools regularly contain undigested food particles, are green, white, brown, rarely yellow, and have a most offensive odor of a putrefactive character. This odor is very far reaching and tenacious, and rather typically present in this condition. The total volume of fecal matter passed is rather large, as most of these babies eat enormously and absorb very little.

The stools seem to be very irritating to the malnourished skin, and we regularly find the buttocks excoriated and red; and bed-sores may develop over the sacrum, occiput, heels, and at times over the ears. The child usually lies in one position, dozing much of the time, always sucking its thumb or fingers, frequently until the skin becomes excoriated, and noticing very little that goes on round about it. If disturbed, or if its fingers are taken out of its mouth, it frets and whines until fed or left alone again to its favorite habit. Some infants whine continuously, and are evidently in persistent discomfort.

Nervous symptoms, misnamed hydrocephalus, may develop; twitchings, rolling of the eyeballs, picking at objects (as the bed-clothes), and even convulsions may occur. The neck may be retracted and stiff.

The tendency of the disease is regularly onward toward a fatal termination. This comes most often from exhaustion with a very low temperature. At other times it is due to a general convulsion, but quite frequently is the result of some intercurrent disease. In the rare cases of recovery improvement is very slow, and months are often taken before the tissues seem to regain their proper tone.

**Diagnosis.**—This depends almost entirely on our ability to exclude all forms of organic disease. In the first place we must be sure that there is no possibility of the presence of active or latent tuberculosis. Of course, the discovery of a local focus of tuberculosis in lymph nodes, lungs, bones, or meninges would at once put us on the right track, but it must be remembered that the lymph nodes often enlarge, and that atelectatic or congested spots often form in the lungs in marasmus, which may be decidedly confusing. Fever ordinarily accompanies any form of tuberculosis, while it is absent in uncomplicated marasmus.

Progressive wasting is not so characteristic of tuberculosis in infancy as is commonly supposed. At that time of life tuberculosis is, in most cases, of the general miliary type, and is quickly fatal, with rapid wasting perhaps, but not with the slowly progressive loss of flesh characteristic of infantile atrophy.

Chronic gastroenteric catarrh has many of the same symptoms as marasmus, and at times the differential diagnosis will be very difficult. The history of the beginning of the two diseases is different, however, and a careful study of the action of the stomach and intestines, together with critical scrutiny of the stools, will aid in the diagnosis.

In hereditary syphilis wasting is often present, but again the history of early coryza, rashes on the skin, and mucous patches at the mucocutaneous junction will assist. The effects of treatment with antisyphilitics will be of value here.

**Prognosis.**—Under any circumstances marasmus is a very serious disease. In institutions it is almost invariably fatal. It is possibly less so among the poor and ignorant in their homes, and somewhat less so when occurring among people in better conditions of life. But even here, where directions can and will be intelligently carried out, and everything that is needed can be procured, it is often very difficult to get the infant's nutrition started on the up grade. If once this beginning is made, the cure follows by a very gradual gain in weight week by week, and a slow return of all the tissues to a proper degree of nutrition.

When recovery does occur the infant returns absolutely to normal, and no results of the disease are left behind.

**Treatment.**—The first important point in treating this disease is to change and improve the surroundings in which the baby has been living. For instance, an infant in an institution or hospital that has developed marasmus has many more chances for recovery if sent out into a private home, and often when this home has not all of the so-called best sanitary surroundings, than if kept at the institution with a number of other children. The quiet and the individual care, and the absence of what has been called for want of a better name "hospitalism," serve to combine for better results than continued life in the institution, no matter how carefully that is watched. In a similar way a child in a well-conducted home will often be benefited by a change of air, such as would be found in a different climate.

The infant should be given an abundance of fresh air; should have regular daily massage, bathings, and alcohol rubs, which should be ended by a cool douche to stimulate respiration; its position in bed should be frequently changed to prevent hypostatic congestion; formation of pressure sores; its mouth should be frequently washed out with saturated boric acid solution or other mild antiseptic, to prevent the development of thrush or other form of stomatitis; its diapers should be changed at once, whenever wet or soiled, to save the skin of the buttocks and neighboring parts from irritation, and to prevent the possibility of a further fresh infection through the stool. If thrush, intertrigo, or bed-sores have developed they should be treated in the ordinary manner with great promptness and care, and

any form of complication, be it ever so mild, retards the chances for recovery.

Of greater importance, but not so much so that the above points can in any way be neglected or overlooked (for the greatest attention must be paid to every little detail to accomplish results in this disease), is the condition of the digestive tract and the system of feeding. The digestive canal must be thoroughly cleaned out to remove any possible bacterial poisons or toxin irritants that may be hindering the proper assimilation of the food. Calomel in 0.006 gm. ( $\frac{1}{10}$  grain) doses every hour for ten doses, or a teaspoonful of castor oil, seem to do this most thoroughly. After either has acted, a thorough washing of the colon by means of a soft-rubber catheter, of No. 8 to 10 French, passed high in the rectum, using warm normal salt solution and allowing it to flow in and out until the fluid returns clear, is the next procedure in order. These two therapeutic measures may often be advantageously repeated every three or four days. By these means we often can put the intestinal absorbents in a condition of readiness to take up and carry into the system a properly prepared nutriment, which it is our next effort to offer them.

The actual feeding problem is a very difficult one, and each case must be a law to itself, as no two cases will give the history of the same kind of previous feeding, or of the same results of that feeding. On general principles it will usually be easy to find some palpable error, or more likely errors, in the feeding method in use. The simple fact that the baby has become marantic on a given food is evidence enough of the necessity for a change.

In the rare case where the infant is at the breast a careful study of the mother's milk must be made, and if defective this must be replaced by that of a proper wet-nurse, or supplemented by artificial feedings of some food that supplies the deficiencies found in the breast milk.

In the more common case of the artificially fed infant we will usually find that the demands of the infant's abnormal appetite have been supplied by a very excessive quantity and an altogether too rich quality of food, certainly by one which this particular marantic infant can in no wise digest properly or absorb properly, and the unoxidized remnants of which only act as poisonous irritants and splendid culture media for the large number of intestinal bacteria which are always ready to enact their life processes, much to the detriment of their host. Indeed, too much food is the usual mistake which we must correct. The quantity must be governed by the powers of the intestinal absorbents and not by the seeming demands of the baby's appetite. The baby's weight rather than its age should be considered in deciding on the quantity of food to be administered, and *only* so much given as seems to be properly taken care of by the digestive and absorptive systems.

The rule is, therefore, to begin treatment by a minimum quantity and dilute quality of food, very cautiously adding to both as the story of the stools and the baby's weight tell us the time is ready. The point to be sure of is that complete assimilation of what is given is taking place before adding more. This food should be, to begin with, carefully



modified cows' milk, as fresh and free from bacterial or other contamination as can possibly be obtained. It had better be fed unless the season of the year or other accidental cause should decide contraindicate. As diluent, the consensus of opinion seems to be decidedly in favor of cereal decoctions as especially valuable in disease.

The casein percentage should be kept very low, but the soluble proteid percentage can be proportionately raised by the use of whey mixtures, as will be found in other parts of this work. (See p. 160.) It must be remembered that the proteids are our best tissue builders and tentatively more and more must be gradually added to the food as the digestion can take care of them, or gain in weight will be very slow. The total proteids should be from 1 to 1.50 per cent. When a satisfactory food is not continued for too long a time.

The fats are quite likely to be undigested in this disease, and must be also used with great caution and in small amount, until the digestion gradually accommodates itself to their increase, which again must be slow. The fat should be from 0.5 to 2 per cent. The yolk of egg in small quantity may be tried in some cases with advantage.

There seems to be no special necessity for reducing the sugar below the ordinary 6 to 7 per cent., and, as a rule, there seems no advantage in the use of one form of sugar over another. Cane-sugar or lactose may be used indiscriminately unless cane-sugar occasions fermentation. The addition of a few grains of sodium chloride to each feeding seems to be of some value in assisting in the osmosis of the food.

In some cases the use of a wet-nurse until the baby obtains its start on an improved nutrition is an absolute necessity, as often the infants cannot digest any modification of cows' milk.

Gavage may be required in the feeding of babies who are very weak.

In all cases careful study of the character of the stools themselves, of their volume proportionate to the food taken, and of their frequency or infrequency, combined with daily weighings on correct scales, gives us our knowledge of the condition of the digestion and assimilation, which is to guide us in our further dietetic and therapeutic procedure.

Drugs are unimportant in treating marasmus, but may be used as adjuncts in dealing with many of the accompanying symptoms. The use of *nuxvomica* alone, or combined with dilute hydrochloric acid may give a tone and stimulus to the digestive canal which will aid it in the work asked of it. A constipated baby will gain faster than a baby with diarrhea; so we would rather these infants do not have too loose bowels. If this looseness is present and is due to unabsorbed food remnants, correction should be dietetic, but if due to excessive peristalsis a little opium may be of value.

Excessive restlessness and fretfulness and insomnia may be helped by a little bromide or chloral. Vomiting is treated by dietetic measures and often by lavage. The use of iron, cod-liver oil, or other "tonics" is best left until the convalescent stage, when they can be added to the dietetic procedures.



## SECTION VI.

### INFECTIOUS DISEASES.

By ISAAC A. ABT, M.D.; DAVID BOVAIRD, JR., M.D.; D. J. McCARTHY, M.D.;  
MATTHIAS NICOLL, JR., M.D.; JOHN RUHRÄH, M.D.; FLOYD  
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#### CHAPTER XV.

##### TUBERCULOSIS.

##### THE TUBERCLE BACILLUS AND THE TUBERCLE.

By ISAAC A. ABT, M.D.

THE tubercle bacillus appears in the tissues as a short, slender rod, 2 to 5 $\mu$  in length. Most recent investigators believe that it is properly classified with the streptothrices, not with the bacteria, because of its tendency to produce branching forms in culture media. It belongs to a group of acid-proof, alcohol-proof organisms; that is, these organisms when deeply stained with aniline dyes by prolonged immersion, or by heating, do not lose their color on the application of mineral acids or alcohol. In the bodies of the bacilli are often seen both unstained portions (vacuoles) and especially deeply stained (metachromatic) granules. Both of these have been supposed to be spores, but this view has become generally discredited, since tubercle bacilli are killed in a few minutes by a temperature of 70° C. (158° F.). Especially characteristic is their very slow growth on all culture media and their difficult development at high or low temperatures—range 29° to 40° C. (84° to 104° F.). The latter factor makes the organism strictly parasitic; it does not multiply except in the animal body. Dried sputum retains its virulence for from three to four months; sunlight or any of the ordinary antiseptics destroys the bacilli, if sufficiently exposed.

The localization of the tubercle bacillus in the tissues is followed by very characteristic pathological changes. The most important is the production of *tubercles*. These are small nodules, produced by proliferation of connective tissue and a moderate emigration of leukocytes. The nodules in the course of their development undergo caseation in their centres. Microscopically, the changes are exactly alike, no matter

what part of the body may be affected. A fully developed tubercle presents the following appearance: in its centre is an area of necrotic tissue, at the periphery of which a variable number of giant cells are usually found. Each giant cell consists of a large mass of degenerated protoplasm with five to twenty nuclei at its border. The necrotic tissue is surrounded by a wall of epithelioid cells, these in turn by round cells. Beyond the round cells is a layer of mature connective tissue. If this layer is complete, the tubercle is said to be encapsulated, and under the circumstances considered healed. Often lime-salts are deposited in a tubercle; it is then said to be calcified. Bacilli are found in greatest number in young, growing tubercles. In older ones they are usually few in number and appear in the periphery of the necrotic tissue and in the giant cells. If the tubercle attains a large size, as in the lung, the necrotic area becomes very soft and finally liquefies and forms a tuberculous cavity. It is a peculiar fact in the pathology of tubercle that capillaries do not tend to regenerate. Caseation in older lesions may be explained in part by the diminished vascularity of the tissues. The tubercle bacillus and its toxins are the exciting factors in the degeneration. (See Plate VII.)

**Localization of Tubercles.**—No tissue or organ of the body is immune from tuberculous invasion. Bloodvessels are seldom involved; indeed, so rarely have they been found affected that for a long time it was believed that they presented an immunity from infection. The foregoing belief had some foundation in fact, since large vessels may remain free from attack, at times a large bloodvessel being the only uninfected structure in a tuberculous pulmonary cavity. The arteries and veins do not always remain free from infection.

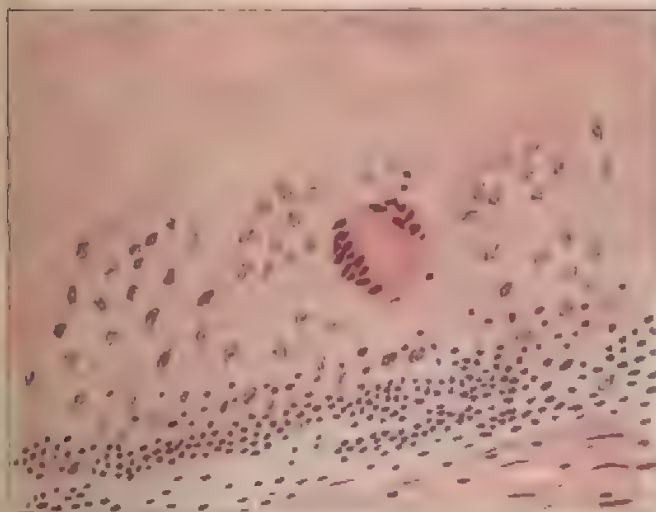
Tuberculosis from neighboring foci may extend into the vessel walls; ultimately, the intima and the blood itself may be invaded by tubercle bacilli. In this manner generalized tuberculosis may originate. The recovery of tubercle bacilli from the circulating blood by our present technique has been successful in very few cases. The thoracic duct is sometimes involved, not so commonly as are the bloodvessels. The infection of the lymph in the thoracic duct usually takes place from the lymph nodes of the abdomen or thorax. If such an infection takes place, a more or less general tuberculosis is inevitable.

Tuberculosis occurs more frequently in the bronchial lymph nodes than in any other organ or tissue. Steiner and Neuritter showed that in 302 autopsies in tuberculous children, the bronchial lymph nodes were involved 275 times (91 per cent.). In the well-known autopsies of Northrup, it is noted that in 125 autopsies the bronchial lymph nodes showed tuberculous changes in every case, irrespective of the cause of death.

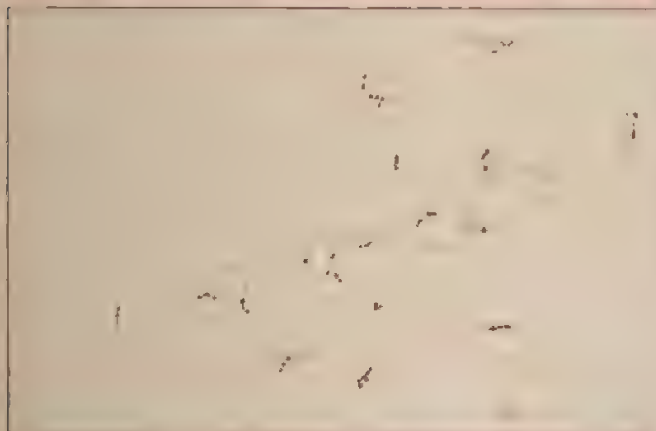
The lungs, pleura, spleen, intestines, liver, and meninges are involved in the order here named. In the genitourinary tract primary tuberculosis is relatively rare. It has been suggested that female children are more rarely attacked than women because of the intact hymen, which acts in obvious ways as a barrier to infection of these parts.

# PLATE VII

Fig. 2

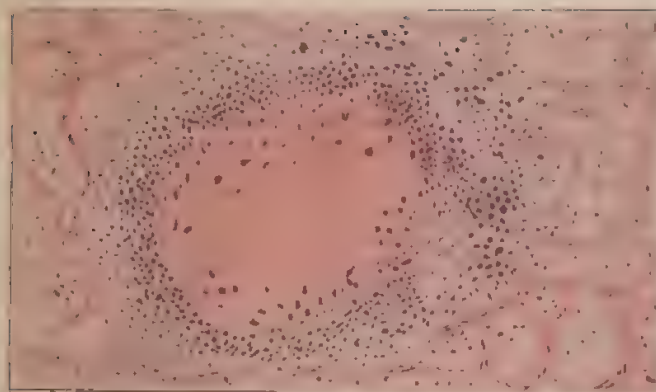


Tuberculous Lymph Node Examined  
with Low Power.



Branching Tubercle Bacilli Grown  
on Glycerin Potato

Fig. 3



Tubercle of the Lung Seen under  
High Power.





Miliary tubercles are frequently found in the genitourinary tract as a manifestation of generalized tuberculosis. Extensive cheesy degeneration is less common than the miliary variety; somewhat more frequent is caseous degeneration in the testicle and Fallopian tubes.

The tuberculous process produces indirectly tissue changes as a result of the toxins produced by the bacilli. Parenchymatous degeneration of the viscera is almost constantly present. Areas of focal necrosis in various organs not specifically tuberculous are observed. The liver shows more or less fatty change, particularly in the pulmonary cases. Amyloid degeneration occurs in the protracted cases of tuberculosis. The spleen and liver show the most striking changes.

**Etiology. Heredity.**—Notwithstanding the frequency of tuberculosis during the first period of life, the existence of fetal and congenital tuberculosis has, in rare instances, been satisfactorily demonstrated. That infection may occur *in utero* is well attested by the case of Schmorl and Birch-Hirschfeld. A pregnant woman died of acute miliary tuberculosis. In the capillaries of the liver of the fetus as well as in the placenta tubercle bacilli were found. Inoculation with portions of the liver and kidneys of the fetus reproduced the tuberculosis.

The relation of paternal tuberculosis to infection of the progeny has been the subject of much discussion. Tubercle bacilli may be found in spermatozoa. They may occur in the semen of men who are suffering from tuberculosis of the genital apparatus; on the other hand, they have also been found in the seminal fluid of men who were known to be tuberculous, but whose genital organs were normal. Notwithstanding these facts, and the frequency with which these organs are involved in men, the transmission of tuberculosis by male inoculation must be considered unproven. Experiments have shown that the male may present active tuberculosis of the genital organs at the time of conception, but the offspring may be born free from the disease.

In the ova of some mammals, as well as in the spermatozoa, tubercle bacilli have been found. Our knowledge concerning the infection of the ovum with tubercle bacilli is, to say the least, not extensive. Virchow has pointed out that ova infected with tubercle bacilli are almost incapable of fecundation. In other words, he believes that the activity of the tubercle bacillus would be fatal to the cell life of the egg. The consensus of opinion favors the view that tuberculosis is very rarely or never conveyed to the offspring by the ovum.

In cases where tuberculosis is actually transmitted from the maternal organism to the fetus, we must presuppose the existence of a tuberculous condition of the placenta. It is not settled as a fact that tubercle bacilli, pre-existing in the organism of the mother, can pass from the maternal to the fetal circulation. In a few recorded cases, where tuberculosis has occurred in the fetus, tuberculous lesions of the placenta have been demonstrated. In the few cases which have been recorded as congenital tuberculosis, and are considered authentic, death has occurred *in utero* or in the first few days of extrauterine life. There are still those who believe that extrauterine infection is not a sufficient cause to explain

the numerous cases of tuberculosis that occur in the first and second years of life. They believe that the disease in many children is congenital and remains latent until roused into activity by some morbid condition which lessens the resistance of the child. In this way they would explain the sudden appearance of tuberculosis after measles and whooping-cough. This view of latent congenital tuberculosis cannot be accepted in the present state of our knowledge; indeed, in view of the accumulated investigations, the general consensus of opinion favors the belief that the large majority of cases of tuberculosis in children is acquired, not congenital, in origin.

*Modes of Infection.*—In man, as in other susceptible animals, the modes of infection are by direct transmission through the excreta, by wounds, or by the ingestion of food derived from other infected animals. Tubercle bacilli are sometimes found in the feces and urine of patients ill with intestinal and genitourinary tuberculosis. In these excreta, however, the bacilli are rapidly rendered inert as a result of the chemical decomposition. Dried sputum, on the other hand, as has been stated, has been found to contain virulent bacilli four months after expectoration. The bacillus occurs almost constantly in the dust of rooms which are occupied by tuberculous patients. This is particularly true of those who cough and raise sputum. Since the bacilli are destroyed by direct sunlight, they are more likely to be found in numbers in dark than in well-lighted rooms. It is a matter of clinical as well as of bacteriological knowledge that the absence of fresh air and sunlight, as well as close confinement with other individuals suffering from tuberculosis, forms the most favorable condition for the transmission of the disease. The most common mode of infection is the inhalation of dust containing virulent organisms. Mouth-to-mouth infection was more common formerly than now; midwives used to blow into the mouths of the newborn infant in order to hasten the first respirations.

The milk of tuberculous mothers is a source of infection. Tubercle bacilli have been found in human milk in cases where tuberculous mastitis was present as well as in those cases where the woman was tuberculous, though the breasts were not the seat of disease. It is believed by many that the foregoing fact accounts for the relative frequency of intestinal tuberculosis in young children. Tubercle bacilli have also been found in cows' milk, and there can be no doubt that in some cases children are infected in this manner. However, there are many who believe that the infection through cows' milk is not as frequent as was at one time supposed. Milk containing tubercle bacilli would naturally produce primary intestinal lesions. Primary intestinal tuberculosis is rare when compared with the enormous frequency of the disease in cattle, the highest statistics giving 7.4 per cent. (Heller), the lowest 0.5 per cent. (Ganghofner) of primary mesenteric or intestinal tuberculosis in children. English statistics, however, are much higher. (See p. 369.) The extensive statistics of Ganghofner show no relation between the occurrence of human tuberculosis and that of mammary tuberculosis of the cattle in the same districts. Th

organisms have also been found in many samples of butter which have proved virulent toward animals. Meat of tuberculous animals has been infective in isolated cases. Since tuberculosis of muscles is extremely rare, this finding may have been due to contamination with tuberculous material contained in other parts of the animal's body.

Infection through wounds is relatively rare in children and has resulted chiefly from vaccination and ritual circumcision. In the latter instance the source of infection is usually the saliva of the operator, who applies his lips to the freshly made wound. This procedure fortunately is rapidly becoming obsolete. The cervical lymph nodes may also be infected through abrasions of the skin of the face, as in eczema.

The bacilli generally enter the human body through the respiratory tract. This manner of invasion most satisfactorily explains the almost universal involvement of the bronchial lymph nodes in young tuberculous subjects. Many maintain that intestinal tuberculosis is almost always caused by the swallowing of sputum which is derived from a primary pulmonary focus. At any rate, intestinal or mesenteric tuberculosis is very rarely found to exist alone. Other organs in remote parts of the body are usually involved before the intestinal or mesenteric infection has taken place.

*Predisposing Factors.*—Food and general hygienic conditions play an important role as predisposing factors to tuberculosis. Tuberculosis is more common in children of the city than in those of the country. The tenement-house districts of a large city are pre-eminently breeding places for tuberculosis. Many infants and young children are housed up all winter in ill-ventilated, filthy rooms. The general resistance is lowered and the invasion by the tubercle bacillus is invited. As has already been stated, some of the infectious diseases, particularly measles and whooping-cough, predispose to tuberculosis. These diseases nearly always cause enlargement of the cervical and bronchial lymph nodes and prepare a soil for tuberculous invasion.

Not every child that inhales tubercle bacilli contracts the disease. The production of the disease depends partly on the number of bacilli inhaled; partly on the susceptibility of the individual. Any acute or chronic disease of the tonsil facilitates the entrance of the bacilli. The tonsils may themselves become the seat of tuberculous lesions, or the bacilli may filter through the tissue of the tonsil into the lymphatics and involve successively various groups of lymph nodes in the neck, thorax, and abdomen, or any other site where lymph nodes are found. Next to the lungs, the tonsils are probably the most common route of entrance. Poorly nourished children, as well as those who are of "lymphatic habit," with large tonsils and adenoids and generally enlarged lymph nodes, are susceptible to infection. The *habitus phthisicus*—that condition which is manifested by narrow, flat chest, and drooping shoulders, with winged scapulæ, prominent *angulus Ludovici*, and weak inspiratory muscles—is more frequently the result than the cause of tuberculosis.

Injuries in some cases have caused the sudden occurrence of tuberculous lesions at the site of injury. It is obvious that a rapid tubercu-

lous invasion could occur only in cases in which tuberculosis existed previously in the body. Tuberculous osteomyelitis and tuberculous meningitis may occur in this way.

*Age of Occurrence.*—As stated before, tuberculosis is rarely found in the first weeks of life; with each succeeding year of life the number of tuberculous individuals increases. In 690 autopsies on children under one year of age, Schwer found tuberculosis in the following number:

Age	Number of cases.	Number tuberculous.	Per cent. tuberculous.
One day to one month . . . . .	268	0	0
One month to two months . . . . .	123	1	0.8
Two months to six months . . . . .	144	15	10.4
Six months to twelve months . . . . .	160	28	17.5

In autopsies on children up to fifteen years of age, Simmonds, Schwer and Bolz found the following number involved at various ages:

Age	Number of cases.	Number tuberculous.	Per cent. tuberculous.
Under one year . . . . .	1433	64	4.5
One to five years . . . . .	781	230	29.3
Five to ten years . . . . .	228	78	35.0
Ten to fifteen years . . . . .	162	56	34.6

Müller believes that children from two to four are the most susceptible; he found this as a result of 500 autopsies. The involvement by years was as follows:

One to five years . . . . .	50.7 per cent.
Six to ten years . . . . .	26.0 "
Eleven to fifteen years . . . . .	23.3 "

Of those under five years, over 80 per cent. were between two and four; only three cases were under one year of age.

*Diagnosis.*—The diagnosis of tuberculosis in the early period of infection is beset with difficulties. The onset of the disease may be insidious and its progress slow. The production of gross tissue changes may be protracted. While it is true that the bacilli may have gained access to the organism and may be active in the production of toxins, it will be noted later that we have not as yet any satisfactory method of recognizing toxic substances of tuberculous origin. The family history should be inquired into, not only on account of the hereditary influence, but also because the presence of tuberculosis in the family suggests the possibility of a house or contact infection. More important than the family history is the information derived from the environment of the patient, particularly as to whether or not there has been any direct exposure to infection. Local tuberculous tissue changes appear somewhat later in the disease; so that, as a matter of fact, what we ordinarily consider as incipient tuberculosis in a clinical way consists in every instance of more or less actually established tuberculous infiltration.

Of the various diagnostic resources, the information derived from finding the tubercle bacilli in excreta, exudates, or tissues is the most positive. In the pulmonary cases bacilli are not usually found in the



sputum until more or less extensive destruction of tissue has occurred. Even in the cases where the sputum is abundant it is not obtainable because infants and children very seldom expectorate. (To obtain sputum from young children for examination the index finger is covered with a strip of gauze and introduced well into the pharynx, so as to irritate the epiglottis; the coughing which occurs as a result of the irritation will frequently cause the expectoration of sputum; this may be caught up on the gauze sponge, diluted in a small quantity of sterile water, and examined for tubercle bacilli.) The examination of other excreta or exudates for tubercle bacilli should be made in appropriate cases, though the results are not always satisfactory. The examination of tuberculous exudates, such as cerebrospinal, pleuritic, ascitic, and other fluids, which always contain very few bacilli, is most effectually made by inoculating the fluids into animals, and at least 10 or 15 cm. of the exudate should be used. Guinea-pigs and rabbits are most suitable for this purpose. The animal may fall sick and die from the disease in two or three weeks, though several months may elapse before a decisive reaction occurs.

Cytodiagnosis, which attempts to distinguish the nature of the exudate by the character of the contained cells, is not altogether reliable. While it is true that in most tuberculous exudates lymphocytes are the predominating elements, non-tuberculous exudates may show the same condition. The examination of exudates is obviously of only limited application. It is evident that tuberculosis of the solid viscera, lymph nodes, etc., is not within the range of this latter method of diagnosis.

The serum diagnosis of tuberculosis devised by Arloing and Courmont has proved of relatively little value. The technique employed is similar to that of the Widal examination for typhoid fever. The homogeneous bouillon culture of tubercle bacilli is used for the test. To this bouillon culture may be added suspected blood serum, or any of the body fluids. If the test is positive in a 1:20 solution, agglutination of the bacilli will occur in about twelve to fifteen hours. Solutions in a strength of 1:5 to 1:50 should be employed for the same test; ordinarily the reaction occurs in from two to six hours. It is believed that the tubercle bacillus is not motile; consequently, the test for motility cannot be applied as in the Widal test. In adults the test is of little value, since many have latent tuberculosis. The test may prove of greater value in children, because latent tuberculosis is relatively less frequent. Agglutination has been found when no tuberculosis was present. On the other hand, tuberculosis has been present and the reaction has proved negative. In miliary tuberculosis particularly, the serum reaction has proved of little value in diagnosis.

Of the various methods for the diagnosis of occult tuberculosis, the most valuable one at present is the injection of tuberculin. Tuberculin should not be injected in patients who are having fever. If the tuberculous patient is running a febrile course, the injection of tuberculin is likely to cause destructive changes in the actively inflamed tuberculous tissues. Again, if the patient is febrile before the injection,

it is difficult or impossible to know whether the rise in temperature is due to the tuberculin or simply a fluctuation in the temperature due to the disease. It is not to be denied that the use of tuberculin has been harmful to some patients. It is possible to change a latent focus into an active one. The most experienced and careful clinicians, however, resort to the use of this injection, and believe that no harm results if small doses are employed. Artificial sera, such as physiological salt solution, have been injected in place of tuberculin. They give reactions in most cases, producing rise of temperature and chill, probably acting in a similar way on the tuberculous tissue as the tuberculin. They are not, however, so reliable as tuberculin, and are probably just as harmful.

Koch has prepared two varieties of tuberculin, one of which is advocated for diagnostic and the other more especially for therapeutic use. The first one is obtained from a culture of the bacilli in alkaline glycerin-bouillon. Of this preparation 1 mg. introduced hypodermically into non-tuberculous individuals produces no effect; in the tuberculous the temperature rises to 39° to 41° C. (102° to 105° F.), usually preceded by a chill, and often with pains in the limbs, nausea, and vomiting, occasionally with cerebral symptoms; at the same time the local findings become marked. In one to four days the reaction disappears and can be made to reappear only with larger doses. Recent observers have advised beginning with smaller doses,  $\frac{1}{10}$  mgm., and increasing to 1 mgm. in cases where the reaction is not sufficiently marked. If conducted in this way the procedure is less dangerous to the patient.

The second tuberculin, which Koch now advocates, especially for purposes of immunization, is obtained from a culture of tubercle bacilli thoroughly dried, ground up in a mortar, and shaken up well in distilled water. The mixture is then centrifugalized and the supernatant fluid is preserved for use by the addition of 20 parts of glycerin in 100. For therapeutic use  $\frac{1}{500}$  mgm. may be injected, and is increased gradually to 1 mg., when the first marked improvement is said to occur. It is not desirable that a reaction of more than 1° C. (1.8° F.) occur as a result of these injections.

#### TUBERCULOUS DIATHESIS.

This condition is frequently described as "scrofula." This is an old term and has a mixed meaning. Properly applied, the term refers to inherited or congenital weakness, or lack of resistance, which is manifested by enlargement of the lymph nodes, particularly in the cervical region; also by anemia and frequently by eczema of the face and eyelids. Cornet has concluded not to abandon the term scrofula because he believes that scrofula includes a larger class of disorders than those which are tuberculous. He considers three forms of scrofula: first, tuberculous, second, the non-tuberculous or pyogenic; third, the mixed forms—a combination of the first two. It is true that in many

so-called cases of scrofulous lymph nodes, tubercle bacilli may be found, or tuberculosis may be produced in animals by inoculation with parts of the infected nodes. On the other hand, in some of the cases the lymph nodes are enlarged as a result of other infections. Probably streptococci and staphylococci of slight virulence may cause lymphadenitis. These organisms may gain access to the lymph nodes through diseased tonsils and lesions of the face.

Laser found among 1216 school children only 137 (11.3 per cent.) free from enlargement of lymph nodes. In the majority of the cases the enlargement occurred after acute infectious diseases, as whooping-cough and measles or from adenoids. In the minority of the cases the nodes were tuberculous. A chronic lymphatic enlargement may be brought about by inflammation of skin and mucous membranes (eczema, catarrh of nose and throat, blepharitis, hypertrophy of the tonsils, ulceration of the gums). A very important factor in the etiology of enlarged lymph nodes is caries of the teeth.

#### TUBERCULOSIS OF THE LYMPH NODES.

Tuberculosis of the lymph nodes occurs in two forms: first, the *localized* form which usually affects single nodes or groups of nodes, and, second, the *generalized*, in which all of the lymph nodes in the body may become tuberculous. In the first group the nodes most often involved are the bronchial, mesenteric, mediastinal, and cervical. In a report of 500 autopsies on children in whom tuberculous lymphadenitis existed, the various nodes were attacked in the order mentioned (Müller):

Bronchial . . . . .	81.7 per cent.
Mesenteric . . . . .	57.1 "
Mediastinal . . . . .	11.1 "
Cervical . . . . .	8.8 "
Retroperitoneal . . . . .	7.1 "
Portal . . . . .	4.8 "
Epigastric . . . . .	3.2 "
Retronaxillary . . . . .	2.4 "
Inguinal . . . . .	2.4 "

The cervical lymph nodes are most frequently infected from abrasions on the tonsils, in the mouth, or on the face. The bronchial nodes are usually infected through the lungs; sometimes the infection spreads through the lymphatics from the cervical nodes. The mesenteric nodes are usually infected through the intestinal tract. The primary lesion, through which the infection gains access to the lymph nodes, may be insignificant in size. The loss of epithelium or a slight wound is sufficient to permit the passage of tubercle bacilli. The point through which the bacilli enter need not be tuberculous in character. The most careful postmortem examination may fail to trace the path which the infection took. This failure to find the route of infection led to the belief that the involvement of the lymph node was frequently hema-



togenous. It has also been claimed that tubercle bacilli may pass through intact mucous membrane, though recent investigators have shown conclusively that this theory is untenable. Any non-tuberculous inflammatory condition of the mucous membranes increases susceptibility of these structures as well as of the underlying lymph node to tuberculous infection. In that class of cases where a single group of nodes becomes tuberculous they usually grow to a large size and caseate. In all such cases inflammation occurs in the capsule of the node and the surrounding tissue; this is spoken of as *peradenitis*. As a consequence of this inflammation around the node the latter becomes adherent to the surrounding tissue as well as to the other nodes. Sometimes a process of repair occurs; then the necrotic tissue is surrounded by a thick, fibrous capsule, which, in the course of time may become infiltrated with lime-salts. As is well known, nature adopts this method frequently in curing tuberculous foci. In other cases the centre of the node comes liquid, the skin becomes adherent to the inflammatory mass, and the abscess ruptures externally. Upon microscopic examination the tissues of the lymph nodes present the same appearance as tuberculosis in other tissues. At best, only a few tubercle bacilli are found in the infected nodes.

In that variety which I have spoken of as generalized tuberculous lymphadenitis, numerous groups or all of the lymph nodes of the body are involved. In this variety the process usually begins in the neck or thorax and progresses by way of the lymphatics to the axillary, mediastinal, retroperitoneal, mesenteric, and inguinal lymph nodes. When these are examined, tubercles may be identified microscopically or macroscopically.

In this connection another variety of tuberculosis of the lymph nodes should be mentioned. Some of the cases which were formerly described as Hodgkin's disease are a form of *generalized tuberculous lymphadenitis*, which differs from the varieties previously described and stands out by itself as a distinct type, which the work of Reed and also Longcope would tend to show. These nodes may be separate, or they may form adherent masses with one another. They vary greatly in their consistency, some being quite firm, others being almost gelatinous. Upon macroscopic examination they do not show any necrotic areas. The individual nodes vary in size from a bean to a walnut, and coalescent masses may be as large as an orange. Upon microscopic examination these nodes are sometimes found to contain many small typical tubercles surrounded by areas of lymphatic hyperplasia. The lymphatic tissue cannot be differentiated from the normal variety. In most of the cases of this class, tubercles are entirely absent. The connective tissue is greatly increased and it is to be especially noted that small areas of necrosis are found. In these necrotic areas large pale cells appear with a variable number of nuclei from 1 to 5 or more. These peculiar cells are not identical with epithelioid cells, and are believed to be characteristic of tuberculosis (Sternberg). Sometimes true giant cells are found. It is extremely difficult to find tubercle



bacilli in this variety of tuberculosis of the lymph nodes, and the real nature of the disease often remains entirely unknown, or until pieces of tissue have been injected into animals. This variety of tuberculous adenitis does not caseate. Macroscopic areas of necrosis remain absent as a rule, notwithstanding their progressive enlargement for years. The similarity between this type of tuberculosis of the lymph nodes and that which occurs in cattle has recently led some writers to express the belief that bovine tubercle bacilli were the infective agents in these nodes. The belief is general, though not unanimous at the present time, that the tubercle bacilli of men and cattle are of the same species.

**Symptomatology.**—Tuberculosis of the lymph nodes, as a rule, has no decided influence on the general health. In a small number of cases constitutional disturbances are present. Sometimes the patients become anemic, lose in weight, and complain of loss of appetite. Fever is not the rule in uncomplicated cases; in the pseudoleukemic cases the temperature may reach  $40^{\circ}\text{C}$ . ( $104^{\circ}\text{F}$ ). If fever occurs in the other cases it is usually due to a mixed infection with pus organisms or the existence of a tuberculous process in other organs, most commonly the lungs. As a general rule, the nodes are painless. In those rare cases where pain does occur, it may be explained by the acute inflammatory changes within the nodes; or it may be the result of compression or the encroachment of nerves within the inflammatory mass; thence the neuralgias which occur in cervical adenitis. Exceptionally, neighboring organs, like the esophagus or trachea, may be pressed upon; 26 to 28 per cent. of patients suffering from adenitis have at the same time pulmonary tuberculosis; from the latter disease many die. Less frequent, though by no means rare, are combinations of tuberculous adenitis with tuberculous affections of the bones and joints.

Mixed infection with pyogenic organisms occurs relatively frequently; this causes acute suppuration of the node and an abscess around it. The nature of such an abscess is frequently determined at the operation, when it is found that the abscess contains, in addition to the pus, caseous material. The suppurative process may eliminate the tuberculous tissue, and in this way nature brings about a spontaneous cure. Recovery from tuberculous lymph nodes may occur in every stage of the disease; in hyperplastic lymph nodes connective-tissue proliferation may cause scar tissue and recovery. Encapsulation by connective tissue or calcium salts are methods of cure, which have already been referred to.

**Cervical Lymph Nodes.**—The clinical course is manifold; at one time one has to do with a medium-sized movable tumor in the submaxillary region; at another time fistulae and ulcers over both sides of the neck cover large masses of lymph nodes. A solitary lymph node may be involved, or the nodes which constitute a group may coalesce to form a large tumor mass, or the infection may extend from one group to a neighboring group until several are involved. Sometimes these various collections may coalesce to form a large tumor mass. This affection is often bilateral (Fig. 70).

Sometimes the capsule of the nodes is involved in the inflammation

going on within. This leads to thickening as well as adhesion to the neighboring organs. On account of the connective-tissue growth the nodes become fixed and immovable. The mass becomes adherent to the skin. The skin becomes edematous, tense, and discolored; the affected area prominent and gradually thins out; eventually it perforates and the abscess empties itself; a fistula remains, which leads into the abscess cavity. If the process continues for a longer time

FIG. 70



Tuberculosis of cervical and axillary lymph nodes in an eight-year-old boy.

ulcerations of the skin persist and granulations appear at the opening. In other cases, where the nodes lie more deeply, the abscess may pass between the layers of the fascia or along the sheaths of the great vessels and perforate the skin at some distance—over the clavicle or at the sternal notch, even over the anterior surface of the thorax. In these cases persistent fistulous tracts remain.

*Bronchial Lymph Nodes.*—The bronchial nodes may be enlarged without causing pressure symptoms; on the other hand, serious dis-

turbances may be produced. Pressure on the trachea and bronchi causes narrowing of the air passages, resulting in dyspnea. Cough is an early symptom of pressure. It is frequently paroxysmal in character and may resemble the cough of pertussis, except that there is no crowing inspiration. The paroxysms may be violent and exhausting, ending in vomiting. As the result of the pressure of the mass of nodes, secondary tracheitis, or tracheobronchitis, may be produced. If this occurs, the lumen of the bronchi is narrowed still more. The attack of coughing is at times more frequent at night; the dyspnea is of an asthmatic type, greater on expiration than on inspiration; the sleep is in consequence often restless; dyspnea on even slight exertion occurs. In some cases sudden death has occurred where enlarged bronchial lymph nodes had not been suspected. Upon autopsy it has been shown that a mass of bronchial nodes narrowed the lumen of the bronchi, and in most cases complete closure occurred as a result of a bronchitis secondary to whooping-cough or measles. In other cases, where a node has undergone caseous degeneration, the mass has ruptured into the bronchi, and relief has been obtained from the pressure symptoms, though very soon an acute miliary tuberculosis of the pulmonary type has appeared. The recurrent laryngeal or pneumogastric nerves may be pressed upon or may be involved in adhesions in the perinodular connective tissue. In these cases the symptoms are referred to the larynx or stomach. The cough is hacking and hoarse without expectoration; the voice becomes harsh on account of the paralysis of the vocal cords, or aphonia may occur. Compression of the esophagus, lungs, or other viscera sometimes occurs, but symptoms from this source are rare. There may be pressure on the superior vena cava, in which case cyanosis and edema of the head and upper extremities with enlargement of the superficial veins of the thorax would be the most prominent symptoms. Frenz reported two very unusual cases, in which the large mass of tuberculous bronchial node tissue escaped from its capsule and ruptured into a bronchus. Once in a bronchus, it acted as a foreign body. The patient in attempting to dislodge this tissue from the bronchus succeeded in forcing it into the larynx, where it caused death by asphyxia.

Physical signs of tuberculous bronchial nodes are not always discovered by our methods of physical examination. When it is found that the supraclavicular nodes are larger than the cervical nodes, and no other cause for their enlargement is found, it may be assumed that the bronchial lymph nodes are likewise affected. There is a direct connection between the tracheobronchial nodes and the cervical ones. From this we would expect both to be involved from one infection. Hoffmann has observed that in cases of tuberculous bronchial nodes enlarged lymph nodes may often be felt at the sternal notch, if the head be bent forward. If the lymph nodes are sufficiently large, dulness may be elicited by percussing over the sternum, particularly over the manubrium. If this dulness extends laterally on either side of the bone, it is a sign of some value. Dulness over the sternum may be found



also in enlarged thymus, though in this case the dulness does not extend as a rule, beyond the lateral margins of the bone. The lungs over these nodes in front, and a resonant percussion note may be obtained even if the nodes are considerably involved. It has been suggested that interscapular dulness was of diagnostic value, but the amount of overlying lung tissue is greater here than in front, so that dulness is rarely obtained in the interscapular region. But, as Hall points out, it depends upon the size of the diseased nodes than upon all other factors.

If there be considerable pressure over one bronchus, a difference in the breath sounds of the two sides may result. Owing to the anatomical position of the right bronchus

roughened breathing of this side may be interpreted cautiously; nevertheless any great difference in the breath sounds between the two sides should be carefully noted. Bronchovesicular breathing on the left side, with prolonged and harsh respiration, is always suggestive. Extreme compression of one of the primary bronchi may cause a diminution of breath sounds. Rilliet and Barthelemy believe that enlarged bronchial lymph nodes may at times conduct the tubular breathing to the surface, even though there be no consolidation of the intervening lung. A venous hum is sometimes heard over the manubrium. East Smith pointed out that if a child is in a recumbent position with head thrown back, a venous murmur occurs and disappears again when the head is flexed. It is believed that by extending the head on the neck the nodes are brought close to the sternum and in this manner cause compression of the left innominate vein. Smith believed that this was diagnostic of enlarged bronchial lymph nodes. He thought that a persistently enlarged thymus or any other tumor would not be likely to cause



Disseminated tuberculosis of the lymph nodes in a fourteen-year-old boy.

symptom (Fig. 71). More recent observations have shown, however, that this venous hum is sometimes heard in children without any disease whatever in the bronchial nodes. Petruschky has pointed out that these patients frequently suffer from spinalgia; he considers this a frequent and important phenomenon. He believes this sign is present in about 90 per cent. of all cases. Some of the vertebrae are more distinctly tender than others. The vertebrae involved naturally depend upon the location of the tuberculous nodes.



*Generalized Tuberculous Lymphadenitis.*—This form of tuberculous lymphadenitis presents many of the same symptoms as Hodgkin's disease, with which it was confounded until recently. There is usually more or less wasting with anemia of a secondary type. Leukopenia is more frequent than an increase of the leukocytes. Fever occurs as a rule, which varies greatly in its course. The chronic intermittent fever, which Elstein described as being associated with pseudoleukemia, is also found with generalized tuberculous lymphadenitis. This symptom is characterized by periods of remittent fever, lasting seven to ten days, alternating with periods of apyrexia of like duration.

The striking features of the clinical picture are the large masses of lymph nodes which appear in the course of the first few months. The cervical nodes are first in evidence, later the axillary, and finally the inguinal. Later on the nodes in the thorax and abdomen also enlarge, the infection progressing rapidly downward from the neck and involving successively the mediastinal, bronchial, retroperitoneal, and iliac lymph nodes. The last are often palpated as large intra-abdominal tumors. The nodes are usually firm and freely movable; they do not coalesce with neighboring nodes. At times they are soft. They may become firmly fixed. In some cases they necrose and break through the overlying skin. The growth is usually continuous; there may, however, be short periods during which these nodes remain stationary. Or, as a result of local medication they may diminish slightly in size. (The cases terminate fatally, usually as a result of some intercurrent acute infection.) Other symptoms depend on pressure of the enlarged nodes on neighboring structures, and also on the increasing cachexia.

*Diagnosis.*—The diagnosis of superficial tuberculous nodes is comparatively simple. The most important points are the persistence, after the presumable cause of their enlargement has disappeared, absence of pain and tenderness, and the tendency to form abscesses with fistulae which heal slowly. A history of tuberculosis in the family and its presence in other parts of the body are also of value in the diagnosis. Actinomycotic processes are differentiated by the discovery of ray fungi in the pus; syphilis, by the presence of other specific lesions and the results of antisiphilitic treatment. Nodes enlarged as the result of a mild pyogenic infection must also be distinguished from tuberculous lymph nodes. When all other measures fail, a node may be excised and the diagnosis established by histological examination or inoculation into animals.

The diagnosis of the condition within the interior of the nodes is often difficult or impossible. Sometimes the consistence of the nodes is so soft that fluctuation is suspected; at other times an abscess in the centre of a node is not suspected, on account of the relative thickness of the surrounding scar tissue; the extent of the disease, too, cannot always be determined. The surgeon is frequently surprised to find that the enlarged nodes are more numerous and extend more deeply than superficial examination gave reason to suppose.

The diagnosis of tuberculous bronchial nodes presents great diffi-

culty at times. Early in the course of the affection the diagnosis is often impossible. The most important symptoms upon which diagnosis may be based are pressure symptoms—especially the asthmatic type of inspiration. The signs of Eustace Smith and Petruschky offer at least corroborative evidence. An absolute and relative lymphocytosis, as suggested by Friedländer, may prove of some value. Dulness over the superior part of the sternum or in the intrascapular region, with a hoarse cough or aphonia, pressure symptoms on the vagus, the blood-vessels, esophagus, tracheobronchi, and lungs, are of great importance, but are found only in the latter stages of the disease.

The diagnosis of tuberculous nodes in the mesentery or retroperitoneum depends chiefly on the pressure symptoms which are produced. Sometimes they can be elicited by abdominal examination or by bimanual examination per rectum.

General tuberculous lymphadenitis must be distinguished from leukemia, lymphosarcoma, pseudoleukemia, and syphilis. Leukemia is easily excluded by an examination of the blood. Lymphosarcoma usually begins in the mediastinal or retroperitoneal nodes. These nodes have already grown to considerable size before the lymph nodes in other regions are in evidence. In physical examination of these cases, during the first stage of the disease, dulness may be elicited over the sternum and under the clavicles for a considerable distance. This is due to the fact that the lymphosarcoma tends to infiltrate the tissue surrounding the nodes, particularly the lungs and the pleura. This may be observed when the nodes in the neck and groin are still insignificant in size. The differentiation of tuberculous lymphadenitis from pseudoleukemia can be made only by microscopic examination of the nodes, though the presence of fever and tuberculous lesions in other parts of the body speak in favor of the diagnosis of tuberculosis. The tuberculin test may be needed.

**Prognosis.**—The prognosis is always serious. It depends upon the form of the lymph-node tuberculosis, the age, and the constitution of the patient. The principal danger is of the disease becoming general. This may occur in the following ways: 1. Extension of the disease along the lymphatic system (pseudoleukemic form). 2. Extension through the vascular system, as where a caseous node breaks into the jugular vein—sometimes no cause for the vascular distribution can be determined. General acute tuberculosis is sometimes observed after operations, particularly where large vessels have been opened. 3. A pulmonary tuberculosis may develop, which leads rapidly to death. 4. The development of tuberculosis in other organs, bones, joints, and meninges.

In general, tuberculosis of the cervical lymph nodes is the form of the disease that remains localized for the longest time. Whether the cases will remain local or become general cannot be foretold; there are benign and malignant cases. In the chronic cases amyloid degeneration leading to death may occur. Spontaneous cure may occur in any of the stages. (This has already been referred to.) The tendency to spon-

aneous cure, however, is so variable that it is questionable if one should depend upon it in any individual case. The tuberculous focus is a menace to the individual, and for this reason should be treated early.

#### DISSEMINATED MILIARY TUBERCULOSIS.

Two factors are essential in the production of disseminated tuberculosis: first, the presence of an old tuberculous focus in some part of the body, and, second, the involvement in this focus of some part of the blood or lymph circulation. If these two factors are present, provided that the tubercle bacilli have gained access to the circulating lymph or blood, numerous metastatic foci of tubercles may spring up simultaneously in different parts of the body. Any of the blood or lymph vessels may form the point of entrance. Arteries were formerly believed to enjoy a special immunity from tuberculous infection; more recently this has been found to be an error, since it has been shown that tubercles occur on the intima of both arteries and veins, usually as a result of direct infection from an overlying focus. The thoracic duct may be the seat of tuberculous lesions, the infection being conveyed to it by the lymphatic vessels coming from the retroperitoneal or mediastinal lymph nodes. Particles of caseous material sometimes gain access to the thoracic duct, and these particles may be carried into the subclavian vein, eventually into the pulmonary circulation, and a consequent pulmonary infection results. It has not infrequently happened that tubercle bacilli have gained access to the general circulation during an operation on some tuberculous lesion. This has occurred most frequently in the operations on tuberculous nodes of the neck, resections of the joints, and operations for tuberculous osteomyelitis. Large doses of tuberculin, particularly during the first era of its use, produced generalized tuberculosis, inasmuch as latent foci were rendered active. A case of disseminated tuberculosis, when at its height, may involve any or all of the organs of the body.

**Pathology.**—Miliary tubercles found in various parts of the body tend to keep pace with one another so far as their growth is concerned. Comparing the tubercles in a portion of an organ with another portion of the same organ, they seem to be about of the same age. This holds good, too, when the tubercles in one organ are compared with the tubercles in another organ. Microscopically, they do not show any differences from the tubercles which are found in localized tuberculosis, with the exception that the fibrous capsule about the miliary tubercle is very thin and free from lime-salts and the tubercle does not tend to liquefy in its centre. This is undoubtedly due to the rapid course which the disease usually pursues. If it is subacute or chronic, the individual tubercles may attain a large size. As a rule, the tubercles are small—about the size of a millet-seed; hence the term "miliary tuberculosis." The individual tubercles are firm in consistency; they are normally of a gray color; though in the lungs, spleen, and the liver



they sometimes appear yellowish. As a rule, the oldest and largest tubercles are found in the lung, and the middle and lower portions are most affected. The external surface is dark red, granular in appearance, and the examining finger perceives little hard masses which feel like bird-shot. On cut-section the lungs are bloody and contain little air. The tubercles are very numerous, and about each one is a small, somewhat granular, dark area which is in the nature of a pneumonic infiltration. Careful investigation often reveals small tubercles on the intima of the veins. The liver, spleen, and kidneys, upon careful examination, show numerous miliary tubercles. In some cases tubercles of liver and kidneys are most distinct in the capsule. Their presence in the organ is elicited with some difficulty on account of the parenchymatous degeneration. The spleen is always enlarged; the liver and kidneys also are usually increased in size. This is because of the parenchymatous degeneration occurring in these organs.

Tubercles are very frequently found in the choroid; it has been estimated in 75 per cent. of the cases. This is probably due to the extreme vascularity of this tissue.

**Etiology.**—Debilitated conditions, from whatever cause, predispose to the development of miliary tuberculosis in children. The disease is especially frequent after the acute infectious diseases, particularly those which are associated with bronchitis. It should not be considered that these acute infectious diseases act in a direct causal manner. From a study of all the facts it would seem that these acute infectious diseases caused old latent foci of tuberculosis to become active. Poor food, bad hygiene, and malnutrition are undoubtedly predisposing causes. Numerous cases are recorded where children fell ill with miliary tuberculosis after operations or injuries, particularly those which disturbed old or latent tuberculous foci in bones or joints. It is interesting to note, though difficult to explain, the great frequency of this disease in children as compared with adults. In adults affected with tuberculosis the chronic form is the most frequent. This variety is rare in children. On the other hand, miliary tuberculosis is somewhat rare in adults as compared to the chronic form. Carr, who examined 120 cases of tuberculosis in infants, found that 82 were of the disseminated miliary variety. (See Plate VIII.)

**Symptoms.**—Miliary tuberculosis occurs with relative frequency during infancy and childhood. From what has already been said concerning the pathology of this disease, it is clear that the original focus of tuberculosis may have been latent in the organism for a considerable time before the general infection occurred. The infection may have originated from affected bronchial or mesenteric lymph nodes. Probably next in frequency are caseous foci in the lungs; less often than the foregoing is a thickened, pleuritic exudate, tuberculous in nature. Local processes in the bones and joints may cause the general infection. Chronic tuberculous processes of the mucous membranes, respiratory or digestive tract, may lead to disseminated





Miliary Tuberculosis of Spleen.



tuberculosis. In rare instances no primary tuberculous focus can be demonstrated on autopsy. In these cases it must be assumed that general infection has occurred from the exterior, without focal lesion.

Three clinical forms of miliary tuberculosis may be distinguished in children: (1) that variety which for the first part of its course resembles marasmus in infants; (2) the pneumonic, and (3) the typhoid form.

1. *The marantic type* is peculiar to infants. The disease begins insidiously. If these infants are systematically weighed it is observed that they lose in body weight, more in acute, less in protracted cases. All of these children become thin, pale, and weak. At the very first the appetite is undisturbed and the digestive apparatus presents no symptoms. The child may continue in this condition for weeks or months before any manifest symptoms of the disease appear, but the process continues. The marasmus becomes more manifest. As the disease progresses, disturbances of function occur; at times the child shows loss of appetite, at other times the appetite is voracious. Often it presents dyspeptic symptoms like vomiting and diarrhea, or during the course of the disease it may be obstinately constipated. In the same way the mental state varies; the child may be apathetic and fretful, or it may become irritable. The duration of the period of latency depends on the rapidity with which the tubercles grow, their location, and the intensity of the intoxication.

After the disease has persisted for a time, temperature is observed. First there may be evening exacerbation, the temperature may rise to  $37.9^{\circ}$  to  $38.2^{\circ}$  C. ( $100^{\circ}$  to  $100\frac{1}{2}^{\circ}$  F.), or a constant fever may set in, varying from  $37.9^{\circ}$  to  $39^{\circ}$  C. ( $100^{\circ}$  to  $102^{\circ}$  F.). About this time, too, local symptoms may appear. The lung is frequently the first to be involved. Sometimes the infants cough, or there may be heard upon auscultation a few moist rales; or they may have pains in the chest, though the symptoms are strikingly disproportionate to the physical findings. Respirations are frequent, varying from 60 to 90 per minute. Vomiting and diarrhea occur.

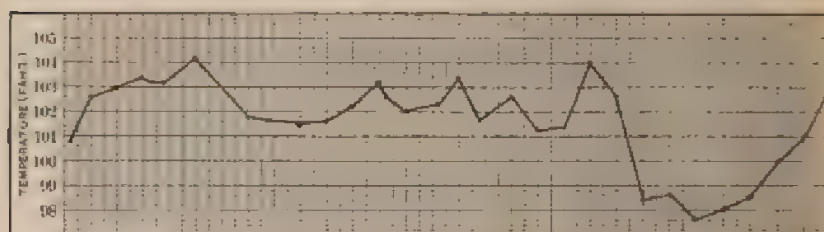
Under the influence of the increasing fever and the general loss in strength, children may die as a result of exhaustion, the disease having run the course of a marasmus. The closing scene may be caused by pulmonary involvement; more rarely, symptoms of meningitis may occur and rapidly terminate the disease. Before death small purpuric spots may appear over the abdomen and extremities.

The physical and clinical findings of this type of the disease remain very meagre until shortly before death. Marasmus may begin in these patients before any gross anatomical changes have occurred in the organs. In some children who have died from infectious diseases no miliary tubercles have been found, though tubercle bacilli were isolated from the tissues. As the disease progresses the tubercles, though they attain their maximum size, are not large enough of themselves to produce physical signs. In the lungs the complicating emphysema makes the detection of small pneumonic areas difficult. The auscultatory

findings are those of bronchopneumonia. The liver and spleen are nearly always enlarged.

2. *The pneumonic form* is most often encountered in children from the second to the fifth year. It may occur as a terminal form in the marantic tuberculosis. It most often follows an attack of acute bronchitis occasioned by whooping-cough or measles. It may be preceded by a prolonged period of malaise or gradual wasting, or its onset may be very abrupt without any prodromata whatever. When it follows an infectious fever, such as measles, the symptoms may arise so early as to make it impossible to distinguish the end of one from the beginning of the other; at other times there is a febrile period of several weeks between the two diseases. The symptoms of the pneumonic form are exactly those of an acute bronchopneumonia. The fever ranges from  $39.5^{\circ}$  to  $40.5^{\circ}$  C. ( $103^{\circ}$  to  $105^{\circ}$  F.), the pulse is accelerated, respirations increase gradually until they reach 70 or 80 per minute, the child is somewhat cyanotic, and extreme dyspnea occurs. The physical findings are very few at the onset; they are those which occur in diffuse bronchitis; later, the respirations become bronchovesicular or bronchial

FIG. 72



Temperature chart in typhoid form of miliary tuberculosis.

in small areas, crepitant rales are heard, and there is more or less impairment of resonance. The most common site of invasion is the middle of the right lung, but the rales are not limited to this area. As a rule, the physical findings are very few until near the termination of the disease. Death occurs in ten days or two weeks in most cases, though at times the disease is more protracted. Symptoms of tuberculous meningitis usually appear at the close of the disease.

3. *The Typhoid Form.*—This form usually appears in children above six years of age. It is preceded by a period of unaccountable wasting. Anemia and digestive disturbances, such as anorexia, nausea, and vomiting, may be prodromal symptoms. Or, fever of a low grade may set in abruptly; it rapidly rises and assumes a continuous type (Fig. 72). The picture closely resembles typhoid fever. Localizing manifestations, as pain and dyspnea, are entirely absent. After ten days or two weeks focal symptoms begin to appear, most often in the lungs. The rales, which were at first diffuse, become most abundant at one or two spots. The respiratory sounds may at a few points gradually assume a bronchial type. The fluctuations in temperature are usually



greater than in typhoid fever. As the time arrives for the child to improve from typhoid, the temperature may abate, but the child continues to waste and becomes cachectic. Usually the case terminates as a tuberculous bronchopneumonia or meningitis.

*Diagnosis.*—The physical findings of the typhoid form are practically none for the first ten days or two weeks; there may be diffuse rales over both lungs; the spleen may or may not be palpable; the urine may contain albumin or a few hyaline casts. Ehrlich's diazo reaction occurs as regularly in miliary tuberculosis as in typhoid, and, therefore, is of no value in the differential diagnosis. A roseola may occur, but not so often as in typhoid fever. Late in the disease the local findings clear up the diagnosis—bronchopneumonia or meningeal manifestations point with great probability to tuberculosis. Periods of temporary improvement followed by relapse and continuous wasting are also characteristic. The Grüber-Widal reaction has been found very rarely.

#### TUBERCULOSIS OF THE LUNGS.

The fact has already been referred to that the bronchial lymph nodes are first to be affected in young infants and children; the tuberculous process extends from these lymph nodes directly to the lungs in most cases. For this reason the middle and lower portions of the lung are the most frequent seat of the tuberculous infection.

*Etiology.*—Tuberculosis of the lungs in children may occur as a diffuse or a localized disease. The diffuse form is usually acute in its course and is almost always the terminal manifestation of a general miliary tuberculosis throughout the body.

In young infants the diffuse form may manifest itself by great wasting. This is spoken of as the marantic variety, and has been already described. Fever may or may not be present; when present it is irregular in type. In children of two to four years the pulmonary symptoms appear after a protracted typhoid-like fever.

Many of the acute infectious diseases, such as pertussis, measles, acute bronchitis or bronchopneumonia (especially if there have been repeated attacks), offer a favorable soil for the development of tuberculosis, particularly of the bronchopneumonic form. The tuberculosis may appear immediately or shortly after one of these infections. Primary tuberculous disease of the bones, skin or genitourinary organs may also give rise to a tuberculous bronchopneumonia.

**Tuberculous Bronchopneumonia.**—Tuberculous bronchopneumonia may be arbitrarily divided according to its duration into three classes: 1. The acute form, lasting less than one month. 2. The subacute form, one to three months. 3. The chronic form.

*Symptomatology.*—The acute variety resembles in its course and physical findings simple bronchopneumonia. The onset is sudden, or it may follow one of the acute infectious diseases already mentioned. The prostration is marked; the cough becomes more and more severe

as the disease progresses. In infants and young children (in whom this type is most common) there is no expectoration. Dyspnea is a marked symptom with 60 to 80 respirations per minute; cyanosis and a weak, rapid pulse are nearly always present; the fever is irregular and relatively low, 100° to 104° F. (37.9° to 40° C.); a persistently high temperature is unusual. The *physical signs* are found in all parts of the lungs; they are unexpectedly meagre, as compared to the marked prostration presented by the child. There may or may not be slight dulness, which is usually pronounced in the upper part of the lower lobes (corresponding to the hilus of the lungs); the vocal fremitus and voice sounds may be exaggerated in the same regions, and small, dry and moist rales are numerous everywhere. The disproportion between the severe symptoms and the slight physical signs is a striking feature of the disease. The course is rapidly and progressively downward, the cough and cyanosis increasing to the end. In the last days symptoms of meningeal involvement may predominate.

The *subacute form* is most frequent in young children. Like the acute form above described it may be part of a disseminated tuberculosis, or it may follow any acute infection of the respiratory tract. If the latter is the case, the onset may be obscured for a time and its course will depend chiefly on the concomitant non-tuberculous bronchopneumonia. When arising as an independent disease or in the course of miliary tuberculosis, it usually appears as an acute bronchitis or bronchopneumonia, and differs from the acute tuberculous form chiefly in its protracted course and in its remissions and exacerbations. The prostration is not so severe; the cyanosis and dyspnea are less marked. When the associated non-tuberculous inflammation subsides a period of remission begins; now, the areas of tuberculous consolidation may be demonstrable, because in this more protracted form they attain a greater size. After the acute infectious diseases the symptoms of tuberculous bronchopneumonia may begin before the child has regained a normal temperature, or there may be a short afebrile interval. The disease begins like an acute bronchitis or an ordinary bronchopneumonia, and cannot be distinguished from such by any signs or symptoms. At the end of two or three weeks the symptoms abate and the signs become less pronounced, but never entirely disappear. There is soon a recurrence, which is more severe than the first attack. Exacerbations alternate with remissions until finally the terminal picture of the acute tuberculous bronchopneumonia is produced. At times the disease may be simply progressive, without remissions and exacerbations and without diminution of the physical signs until the fatal termination. This form differs from the acute form only in being less rapid in its course. The wasting is extreme; lost weight is never regained, although there may be at times interruptions in the progressive emaciation.

The fever depends chiefly on the pulmonary complications, which are, for the most part, simple bronchitis and non-tuberculous bronchopneumonia. The period of remission may be afebrile, or a low range of temperature may persist. During the acute attacks the fever is

irregular, rarely hectic, and not often persistently high. Gastrointestinal symptoms, as anorexia, vomiting, and diarrhea, are usually present; in some cases these symptoms may be due to amyloid changes. Secondary anemia and cachectic edema are frequently present in the last stages of the disease.

In the lungs small areas of dulness are found most often in the upper part of the lower lobes. Bronchophony or bronchial breathing with crepitant and subcrepitant rales are the usual auscultatory findings; sometimes the examination shows the evidence of an acute diffuse bronchitis. The spleen and liver are usually palpable and soft, unless they are the seat of amyloid deposits arising from old tuberculous lesions in other parts of the body; in the latter case the spleen and liver are also palpable, but hard, not soft.

*The chronic tuberculous pneumonia* appears most often in children over five years of age, and approaches the type commonly present in adults. The areas of consolidation due to the tuberculous lesion are extensive, and cavities may form, although from their small size they may be difficult to detect, as they do not always show characteristic signs. The symptoms may appear gradually with slowly increasing severity, or may begin like the acute or subacute forms, continuing with repeated exacerbations and remissions. The findings are those of a bronchitis, pleurisy, lobar pneumonia or bronchopneumonia during the more acute phases of the disease. In the interval they do not entirely disappear; the child remains sickly and anemic. Cachexia is observed in a certain proportion. During this stage of quiescence signs are variable, but remain confined to the chief points of tuberculous involvement. They are found most often in the upper lobes, rarely at the very apices of the lungs; next in frequency in the upper portions of the lower lobes. Externally the changes are best demonstrated anteriorly in the mammary region or posteriorly between the scapulæ. They are characteristic of circumscribed bronchitis, bronchopneumonia or, very rarely, of cavity formation. As stated above, the cavities are seldom large enough to produce definite signs, such as amphoric breathing, large bubbling rales, Skoda's tympany, Wintrich's sign, which is a change in pitch of the percussion note when the mouth is opened and closed, or cracked-pot resonance. Any one or all of these signs may be due to an area of consolidation about a large or dilated bronchus.

During the exacerbations the signs become less characteristic of tuberculosis and correspond to associated inflammatory changes. The condition then becomes that of a diffuse bronchitis with large and small dry and moist rales, or of lobar pneumonia or bronchopneumonia. It is rarely possible by examination during this stage to localize the foci of tuberculosis or to distinguish the disease definitely from a pneumococcus infection.

The duration of the disease varies from months to years. The wasting is progressive, though the symptoms abate in periods. Usually there have been repeated attacks of bronchitis or bronchopneumonia before the true nature of the infection is revealed. The persistence of physical signs during the interval, with occasional rises in temperature,



is suggestive of tuberculosis. Hemoptysis does not occur often in children; when present, the hemorrhage is small in amount and very rarely proves fatal. Death results from miliary tuberculosis, cachexia, simple or bronchopneumonia, or from meningeal tuberculosis.

*Cough* in young children is very seldom accompanied by expectoration, and many means have been devised to obtain sputum for diagnostic purposes. A method of procuring sputum by irritating the epiglottis and catching the mucus on a gauze sponge in the pharynx has already been referred to under Diagnosis of Tuberculosis. Holt recommends the passage of a stomach tube and examination of the mucus attached to it, because a portion of it may be the sputum swallowed by the child.

Tuberculin injections may prove of value in diagnosis in older children, especially during afebrile periods of the chronic form of the disease. As stated before, its use is not devoid of danger.

*Chronic localized tuberculosis*, or phthisis, so frequent in adults, is rarely found in young children, and is not common before the tenth or twelfth year. It does not differ in its pathology or symptoms from the disease in older persons, except that its progress is usually more rapid. The child has frequent "colds," a cough that is rarely entirely absent; it is pale, weak, and presents an increasing cachexia. Fever is usually present, either continuous and of low grade, or during the bronchitis attacks the fever may be of a hectic type. There may or may not be pain in the chest. Foci of dulness are found in the lungs associated with bronchial or bronchovesicular breathing and rales ranging from mucous clicks to large moist sounds. Bronchophony is frequently present. Cavities may occur and are recognized by anphoric breathing, cracked-pot resonance, Wintrich's sign, and the other characteristic findings. The apices of the upper lobes and the bases of the lower are usually spared. Perforation of the lung with production of a pyopneumothorax is rare in children, as are profuse hemorrhages, though the latter do occur.

#### TREATMENT OF TUBERCULOSIS.

*Prophylaxis.*—It has already been sufficiently emphasized that contact infection is the most prolific source of the spread of tuberculosis. For this reason it is of prime importance that healthy individuals should avoid, as far as possible, contact with those who are tuberculous, particularly those who are suffering from an active pulmonary form. A healthy individual should not occupy the same sleeping apartment with one who is suffering from tuberculosis. Lactation is absolutely contraindicated in cases where the mother is suffering from tuberculosis. This is true whether the mammary glands are involved or not. The best possible hygienic conditions should be provided. The food should be most carefully adapted to the varying needs of the mother and child. In families where a predisposition to tuberculosis exists, every possible precaution should be taken to prevent the occurrence of the acute infec-



cious diseases, particularly those which are associated with secondary bronchitis. Wherever it is possible, precaution should be taken to prevent attacks of primary bronchitis. This latter may be sometimes, though not always, accomplished by proper ventilation, avoidance of irritating dust in-doors and out-doors, and by prompt and early attention to the slight catarrhal infections, which frequently proceed downward, causing successively pharyngitis, laryngitis, and bronchitis. The enlarged tonsils and adenoids or other affections causing obstruction to nasal breathing should receive prompt treatment. Life in the open air, frequent bathing, followed by cold or tepid sponging, increases the resistance against infection. Open-air exercise which is directed particularly to the development of the thorax and to the expansion of the lungs is also of value in preventing infection. Those children who are under weight or undersized should be encouraged to take an abundance of nutritious food, especially fat, and should be given cod-liver oil, and if indicated, stomachics or iron tonics for the purpose of improving the general health.

The use of milk or meat from tuberculous animals should be avoided, if this is possible. If the source of the milk used is not known, heating at 65° C. (149° F.) for fifteen minutes is effective in destroying tubercle bacilli contained therein (Theobald Smith). The immunization of cattle by Behring's method of injecting increasing doses of tubercle bacilli derived from man will probably prove of value in removing the danger of bovine infection. Behring believes also that it may be of benefit to feed infants on milk from immune cows, and that this procedure may confer upon them a certain degree of immunity.

The sputum and other excreta of tuberculous individuals should be disinfected. The sputum is not only dangerous to others, but to the patient as well, since it subjects him to the possibility of reinfection. Nor should the sputum be swallowed, as this is so often the cause of intestinal tuberculosis. In infants this danger cannot be avoided.

*Therapeutics.*—There is no specific treatment. Favorable results have been published from the use of Koch's new tuberculin. The tuberculin, however, is not at all suitable for advanced cases.

The best results are obtained by fortifying the general health and relieving the symptoms as they arise. For the first purpose every effort should be made to increase the body weight. This can be best accomplished by an abundance of wholesome, easily digested food and by the use of cod-liver oil. It should not be given, however, if its administration produces gastric disorders or diminishes the appetite. Other oils and fats may be administered as substitutes. The syrup of the iodide of iron and Fowler's solution are extensively used for their tonic effects.

Changes of climate have proven of value not only in cases of pulmonary tuberculosis, but also in others of long standing, especially of the bones, glands, and lymph nodes. High and dry air is most strongly recommended when the lungs and bones are involved, while the sea air in a warm, equable climate appears best for cases of disease of the lymph nodes.

For those individuals who cannot remain away permanently, it is not advisable that a too radical change in climate be made. For instance, a sojourn of six weeks during January and February in California, and then a return to the severe March weather of the Middle West or East will not accomplish good results. Too great differences in climatic conditions are likely to cause reinfection and rapid progress of the disease. The most desirable high climates are those of Arizona, New Mexico, and Colorado. The coasts of Florida and Texas are warm and moist. For moderate change and moderate elevation, good results are obtained in the Adirondacks and Catskills. The region to be sought depends on the season and the climate of the country in which the disease is being treated. Extreme changes should be avoided. In the summer, mountain air should be selected. In winter, a southern region is chosen by preference. Sunshine is always to be sought for.

Rest in bed is essential in the treatment of cases with a temperature of more than  $37.9^{\circ}\text{C}$ . ( $100^{\circ}\text{F}$ .). Otherwise, moderate exercise is preferable. The room in which the patient remains should have all the windows open and as much sunlight as possible. Many sanatoria are so arranged that the patients with fever can spend their entire days and nights in the open air.

**Treatment of Lymphadenitis.**—The prophylactic and general hygienic considerations which have already been discussed apply to the treatment of tuberculous lymphadenitis. It cannot be too frequently emphasized that children suffering from local tuberculosis should have an abundance of fresh air and sunshine throughout the whole year. Children with tuberculous nodes frequently show improvement if they are sent to the seashore for a protracted stay, or to the country for the summer. The nutrition should be maintained, and as far as is compatible with the digestive functions, they should be rather overfed than underfed. Internally, the various tonics are indicated—cod-liver oil and the syrup of the iodide of iron are the most valuable. Fowler's solution, preparations like the albuminate or peptonate of iron, syrup of hydriodic acid aid the general nutrition, increase the resistance of the patient, and sometimes seem to cause an involution of the nodes. If the nodes are localized, as in the cervical region, various external applications, such as mercurial ointment, or the iodine preparations, as the tincture and the compound ointment, have been recommended. These external applications have no therapeutic value and their use should be discouraged. In the treatment of tuberculous nodes which lie superficially the x-ray treatment has been advised, and has undoubtedly been successful in some cases. In that form of tuberculosis of the lymph nodes which closely resembles pseudoleukemia, and which has been alluded to as generalized tuberculosis of the lymph nodes, the same method of treatment should be followed as has previously been outlined. In addition, arsenic, as in Fowler's solution, may be employed in increasing doses, by mouth or hypodermically. The x-ray treatment is recommended also in this form of tuberculosis. In this latter class of cases extirpation of the nodes does more harm

man good. The nodes recur after the operation and the growth is more rapid after the extirpation than before. In the local tuberculosis of lymph nodes, which shows a tendency to increase, surgical intervention is indicated, but all such cases should have the benefit of out-of-door life before operation is insisted upon.

#### **TUBERCULOSIS OF THE INTESTINE AND MESENTERIC LYMPH NODES.**

By DAVID BOVAIRD, Jr., M.D.

Tuberculosis of the intestine and mesenteric lymph nodes practically always occur together and they are therefore considered in conjunction.

**Etiology.**—Tuberculosis of the alimentary tract may possibly be produced by the lodgement of bacilli floating in the blood stream, the primary focus being elsewhere in the body, but such spread of the disease appears to be relatively rare. In the great majority of cases intestinal tuberculosis is produced by bacilli that have been swallowed. These bacilli may come from old tuberculous processes in the lungs just as undoubtedly happens in adult intestinal tuberculosis, or they may be taken into the body with some of the food, the first tuberculous lesions being produced in the intestinal tract. In the first case we have a secondary, in the second a primary intestinal tuberculosis. For many years a great deal of interest has centred about the question of the frequency of such primary intestinal tuberculosis and its causation by the milk of tuberculous cattle. The furore created by Koch's declaration that human and bovine tuberculosis are separate and different diseases, and that bovine tuberculosis cannot be transmitted to man, and *vice versa*, is still fresh in our minds. Both before and since the time of Koch's address many papers dealing with the question of primary intestinal tuberculosis have been published and certain very discordant results are reported. In 125 autopsies on tuberculous children I found just 2 cases of apparently primary intestinal lesion, and in a total of 300 cases from New York, this number including also the observations of Holt and Northrup, there were 5 cases of such primary intestinal infection, a little more than 1.3 per cent. With these figures those of German and French observers fairly well agree. English statistics, however, present a radical difference. In 748 autopsies on tuberculous children, collected from English literature, there were 136, or 18 per cent., primary intestinal infections. In English writings the percentage of such cases is generally stated to be as high as 28 to 30 per cent. It is very difficult to reconcile these figures. It cannot be done on the basis of proportionate variation in the amount of tuberculosis in cattle, but the fact cannot be doubted that conditions prevail in England which are radically different from those in America.

Investigations have shown that there must be abundant opportunities for infection from the milk of tuberculous cattle, tubercle bacilli having



been found in as high as 25 per cent. of samples of dairy milk supplied for use in cities.

It should not be forgotten that there are other ways in which children can receive and swallow tubercle bacilli. Especially is it possible that children living in homes with tuberculous adults should be infected by kissing, or that the child's hands become infected in playing about the floor and the bacilli be in this way carried to the mouth. It has been demonstrated that tubercle bacilli may be found under the nails of children, even when there is no tuberculosis in the home.

Doubtless but few of the bacilli swallowed reach the intestine in a condition to do harm, otherwise intestinal tuberculosis would become vastly more common.

**Pathology.**—Tuberculosis of the intestine and mesenteric lymph nodes is nearly always part of a general tuberculosis, and the tuberculous lesions are found in the bronchial nodes and lungs, the liver, spleen, etc. Intestinal lesions are found in a considerable percentage of all cases of tuberculosis in children. In my series of 125 cases there were intestinal lesions in 36. These lesions are found mainly in the small intestine, but are found in the large intestine also. In the earliest stage the lesions are miliary tubercles; small, pale-yellow grains, about a line in diameter; they may be felt. They are usually found first in Peyer's patches (Figs. 73 and 74). There may be only a few or great numbers of them. They quickly increase in size and then break down, forming small round ulcers with soft edges, showing very little or no induration, and fairly definite outlines; the base is covered with granulations. It is usually quite impossible from the character of the ulcers alone to say whether they are tuberculous, typhoid, or simple. Later, the ulcers fuse into one another and form large excavated areas extending transversely to the long axis of the bowel. On the peritoneal surface of the intestine opposite an ulcer of any size we can usually find a number of minute gray or colorless miliary tubercles. In the older ulcers the edges become indurated, and efforts at cicatrization may be seen in a contracted, puckered, peritoneal coat, and filling in of the ulceration with granulations. These ulcers rarely perforate, though the intestine may be so softened that when taken out and washed it may appear riddled with holes. The peritoneum usually presents a more or less general adhesive peritonitis and may be more or less thickly sown with miliary tubercles.

The lesions of the mesenteric nodes may be an apparently simple hyperplasia, or there may be miliary tubercles, or tuberculous nodules, or diffuse caseation and breaking down, so that the nodes are full of thick, pale-greenish pus. Wherever there are tuberculous ulcers of the intestine, we may be quite sure that the mesenteric nodes are tuberculous, even though on section they appear normal. The size of the individual lymph nodes varies from 1 to 3 cm. They may be grouped in masses of considerable size. Holt speaks of a mass the size of a child's head at birth. It is not uncommon to see masses the size of a hen's egg (Fig. 75).

The peritoneum is usually matted together everywhere and full of

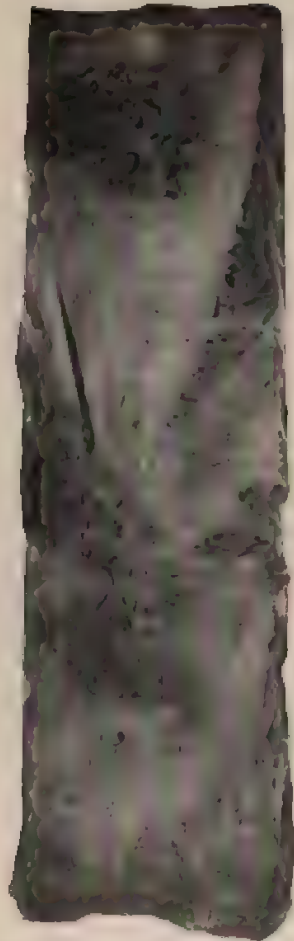


FIG. 73



Tuberculous ulcers of the small intestine. The lowest portion shows the Peyer's patch, just above the ileocecal valve, a favorite seat of ulceration of any kind.

FIG. 74



Hypertrophy of Peyer's patches in the small intestine, with superficial erosions resembling ulcers.

miliary tubercles. It may contain large tuberculous masses, or there may be an exudation of serum into the peritoneum, or any of the conditions described under Tuberculous Peritonitis.

**Symptomatology.**—These are altogether indefinite in the great majority of cases. It is not unusual to find extensive ulceration of the intestine in tuberculous cases in which there have been no intestinal symptoms, and, on the other hand, patients in the last stages of tuberculosis may have severe diarrhea without tuberculous lesions of the gut. In other cases there may be the symptoms of a chronic ileocolitis and the passage of stools containing mucus and blood. The latter is not at all regular or frequent, and the bleeding is more excessive. With involvement of

FIG. 75



A mass of tuberculous lymph nodes; the mass laid open by a mesial section. These nodes lay just at the ileocolic junction, and are the ones most often affected.

the mesenteric lymph nodes and the peritoneum the abdomen is distended and generally tender and tympanitic. In some instances the enlarged nodes can be seen and felt through the thin abdominal walls. With either local or general tuberculosis, the temperature regularly shows more or less elevation. The course of the disease may be very rapid or very slow. Where the intestines and mesenteric nodes alone are involved the course is slow; like that of chronic ileocolitis it may be protracted for months. It is always fatal.

**Diagnosis.**—With a chronic diarrhea attended by fever, distention of the abdomen, and the presence of masses of enlarged lymph nodes the diagnosis may be easy. Likewise, in cases where similar conditions

develop in a child already suffering from tuberculosis. In the primary cases resembling chronic ileocolitis the differentiation has been given under the latter subject (p. 266). In any case the only satisfactory proof of the nature of the lesion is the finding of tubercle bacilli in the mucus of the feces. For evident reasons this may not be easy; repeated examinations may be required.

**Prognosis.**—The disease is fatal sooner or later, death being caused, as a rule, by exhaustion; perforation and hemorrhage have been the immediate causes of death in some cases.

**Treatment.**—This must be on the lines of an ileocolitis. Medicines are of little value. When once we have satisfied ourselves as to the diagnosis the prospect of recovery being practically out of the question, we had best aim to make the patients comfortable.

#### TUBERCULOUS PERITONITIS.

**Etiology.**—In tuberculosis of any part of the body, the intestines, lungs, lymph nodes or bones, or genital organs, it is possible that the peritoneum may be involved. Thus in 125 cases of general tuberculosis, in nearly all of which the lungs were the chief seat of disease, I found the peritoneum involved in 9—i. e., 7 per cent. In 883 cases of tuberculosis Biedert found peritoneal lesions in 18 per cent. These figures are from the results of postmortem examinations and represent the frequency with which peritoneal lesions may be found in children suffering from tuberculosis of other parts. The majority of these cases would not be recognized as cases of tuberculous peritonitis during life. The frequency of clinical peritoneal tuberculosis is quite another matter. This varies greatly in different localities or countries, for reasons which are not clear. There can be no question that tuberculous peritonitis is much more common in Great Britain than in America.

In a single day in the Hospital for Sick Children, Edinburgh, I saw more cases of peritoneal tuberculosis in young children than I had seen in ten years in hospital work in New York City. In America it is certainly a very rare affection, almost never seen in infants, and very rarely in children under the age of seven years.

Peritoneal tuberculosis is practically always secondary. The infection may be carried by the blood stream or by the lymphatics. In the first class the peritonitis is simply part of a general miliary tuberculosis. In the second class the infection travels from some of the neighboring organs, intestine, lungs, spinal column, genital organs, usually by way of the lymph nodes. The primary factor in any case of tuberculous peritonitis is therefore the original infection. In some instances blows or falls on the abdomen seem to have excited the peritoneal disease.

**Morbid Anatomy and Symptomatology.**—The lesions of tuberculous peritonitis are varied, and as the symptoms of the diseases vary with the form of lesion it is best to consider them together.

1. *Miliary Tuberculosis of the Peritoneum.*—This is the form of tuberculosis of the peritoneum regularly met with in cases of general



tuberculosis. The miliary tubercles are few or many; usually there are great numbers. There are generally firm adhesions between the peritoneal coatings of the intestines, and between the intestines, the abdominal wall, and the viscera, so that the peritoneal cavity is practically obliterated. The condition gives rise to no distinctive symptoms and is recognized only at the autopsy.

2. *Miliary Tuberculosis of the Peritoneum with Ascites*.—In this case there is an acute eruption of miliary tubercles with more or less of the manifestations of an acute peritonitis. The peritoneum is congested, cloudy, and may be coated with lymph. There are adhesions between the intestinal coils. There is an abundant effusion of serum, regularly clear, but it may be seropurulent or even bloody. Tuberculous lesions are constantly found in other parts of the body.

The symptoms in this form of peritoneal tuberculosis vary greatly. In some cases the disease begins so acutely as to suggest acute enterocolitis, or intestinal obstruction. There are fever, vomiting, abdominal pain and distention with fluid, and diarrhea or constipation. In other cases the onset is very gradual and insidious and the distention of the abdomen with fluid is the first symptom to attract attention. When the disease is well established, there is regularly some fever, although it may be slight. The digestion is disturbed. There may be occasional vomiting, and the bowels are constipated or loose. The abdomen is then markedly distended, the skin seems thin and pale, the superficial veins are enlarged, and there are the characteristic physical signs of ascites. In some instances the fluid is encapsulated either in the pelvis or flank, and may suggest an ovarian cyst. When the fluid is removed from the abdomen it may be possible to feel some nodules in the peritoneum or enlarged mesenteric nodes; often, however, this is impossible. The fluid reaccumulates rapidly after tapping. The prospect of recovery is usually in keeping with the onset; the acute, severe cases do better than those in which the onset is slow and insidious and the course protracted. Usually do well.

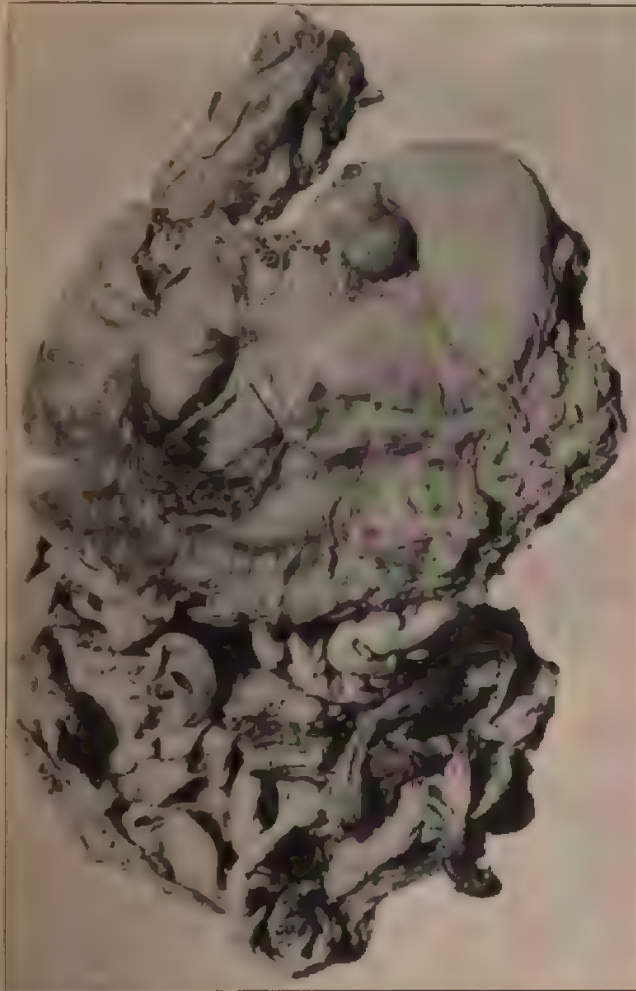
3. *The Caseous or Ulcerative Form*.—In this case there are extensive tuberculous deposits in the peritoneum which go on to caseation. There is usually an abundant effusion of fibrin by which the coils of intestine are matted together and to the various viscera (Figs. 76 and 77). From these adhesions pockets are formed which may be filled with clear serum or thick, tuberculous pus or a brownish fluid. The tuberculous nodules occur in any part of the peritoneum and in the abdominal wall. The process may lead to suppuration and the formation of fistulae, especially often in the neighborhood of the umbilicus. Advanced tuberculous lesions are found in the other viscera, especially the lungs.

The constitutional symptoms in this condition are usually those of a general tuberculosis, with considerable fever; it may be of the hectic type: rapid pulse, rapid respiration, sweating, and marked prostration. The abdominal symptoms consist of indigestion, possibly with vomiting, more or less colicky pain in the abdomen, and constipation or diarrhea. If there are tuberculous ulcers in the intestine, there will occasionally



be blood in the stools. The abdomen is distended, tense; nodules may occasionally be seen beneath the skin. Unless the effusion into the peritoneum is very large the signs are not those of ascites, but of scattered areas of dulness from encysted fluid, with intervening areas of tympany.

FIG. 75



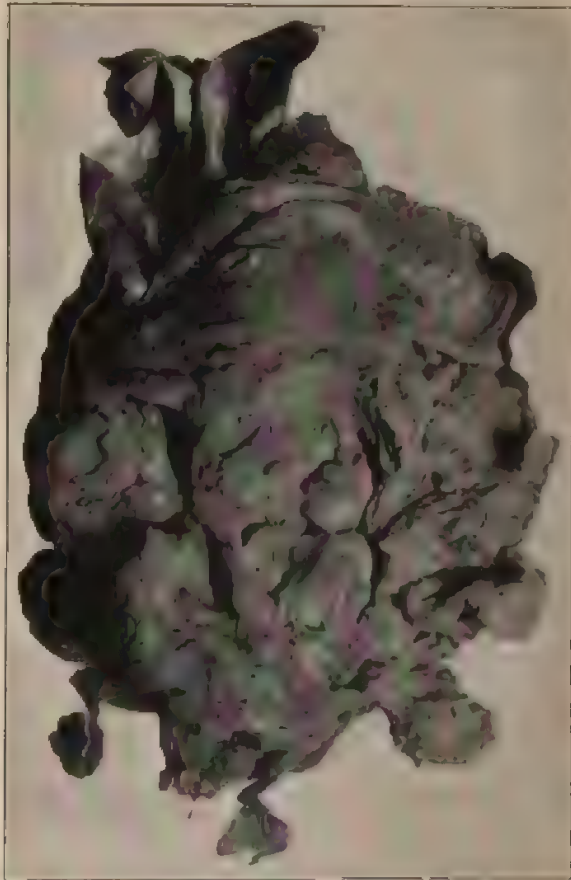
Intestines removed en masse from a case of tuberculous peritonitis. Note the thickened omentum containing tuberculous nodules and the matting together of the intestines.

Often the abdomen has a rather characteristic doughy feel and nodules may be detected here and there in it. In some instances there are fistulae, especially near the umbilicus, discharging characteristic tuberculous pus. In other cases the fistulae may open into the bowel. The course of the disease in this form is steadily progressive, the patients

suffering not alone from the peritoneal but from the general tuberculous lesions. The duration is usually two or three months. The patients die of exhaustion, or from new complications, such as tuberculous meningitis, rarely from the peritoneal process itself.

4. *Fibrous Form.*—In a considerable proportion of all the cases of tuberculous peritonitis there is no effusion of serum or pus, but the

FIG. 77



A mass of intestines from a case of tuberculous peritonitis viewed from behind. Matting of all the tissues and the masses of mesenteric lymph nodes.

tubercles are surrounded by more or less lymph and there is a tendency to cicatrization. In these cases the peritoneum is greatly thickened, dense and firm and full of miliary tubercles, the tubercles being covered with fibrous tissue. The intestines are densely matted together, the peritoneal coating of liver and spleen are greatly thickened and adherent to adjacent parts, and the peritoneal cavity almost completely obliterated. The process is usually general, but may be localized. In some

instances the omentum is particularly affected and is converted into a ridge-like tumor lying across the upper abdomen. In other instances this condition is found in a hernial sac, or about the appendix. It is not infrequently encountered in laparotomies for other conditions.

The symptoms of the fibrous form are very obscure. It may remain entirely latent, to be discovered only at autopsy. Generally the onset is very gradual and insidious. There may be a definite fever, but the temperature is often normal or subnormal. There may be some colicky pains in the abdomen, but these are slight. The bowels may be constipated or loose. The abdomen is usually distended at first from tympanites, later from the peritoneal changes. Sometimes there is ascites, but this is scant. On examination it may be quite impossible to demonstrate any abnormal conditions about the abdomen; usually the abdomen is distended and tympanitic over the greater part. There may be localized areas of dullness, or there may be masses, such as the rolled-up omentum, which can be felt.

Symptoms may be produced by the contraction of the adhesions. Frequent vomiting may be caused by traction on the stomach, or intestinal obstruction from stricture of the intestine, or there may be edema from pressure on the vena porta or vena cava, or albuminuria from involvement of the renal veins.

The course of the disease varies greatly. Spontaneous recovery may occur. The presence of tuberculous lesions in other parts of the body has much to do in determining the final outcome. The duration of the disease varies from a few months to several years.

**Diagnosis.**—In certain cases of miliary tuberculosis of the peritoneum and also of the fibrous variety, the diagnosis is made only at autopsy or operation. In the cases associated with marked changes in the peritoneum and ascites, the diagnosis lies between cirrhosis of the liver and simple chronic peritonitis. Cirrhosis of the liver is in childhood an extremely rare affection, much less frequent than peritoneal tuberculosis. After tapping in cirrhosis we may be able to make out that the liver is abnormally hard or small, and, in syphilitic cases, irregular. Jaundice is more common in cirrhosis; fever belongs to tuberculosis. A study of the cytology of the fluid should help us. In tuberculosis there should be marked preponderance of the mononuclear leukocytes; in cirrhosis we find chiefly endothelial cells. The centrifuged sediment may be examined for bacilli, but the inoculation of a small quantity of the fluid into a guinea-pig or rabbit is a much safer test. Several weeks will, however, be required to determine the question by the latter method. An encysted exudate is always in favor of tuberculosis, likewise the presence of a fistula.

The distinction from simple chronic peritonitis may be even more difficult. This affection is also rarer than tuberculosis of the peritoneum. A good family history, absence of fever and emaciation are in favor of a simple inflammatory process. If ascites is present, the fluid may be tested as suggested above. If the cases are operated upon, a microscopic examination of the fibrous nodules or inoculation of the



exudate may be required, to determine the diagnosis, so closely do the conditions resemble each other.

The ulcerative form of tuberculous peritonitis is easily distinguished by the presence of tuberculous lesions elsewhere and by the marked changes in the peritoneum, the fever, and wasting.

Rectal examination under an anesthetic may be of great help in a doubtful case in enabling one to detect local collections of fluid, masses of enlarged lymph nodes, etc., which might not be accessible to ordinary palpation.

**Prognosis.**—The ulcerative form of tuberculous peritonitis is regularly fatal, although recovery has been known to follow the discharge of an abscess at the navel. In the other varieties the prognosis is fairly good, many of the cases recovering on rational treatment, and many being improved, if not cured, by laparotomy.

**Treatment.**—There has been a great deal of discussion in recent years as to the best treatment for tuberculous peritonitis, and the question is not yet settled. Treatment may be classed as either medical or surgical.

**Medical Treatment.**—Fresh air, quiet, and good nursing are of prime importance. The patients are kept in bed as long as there are active symptoms of disease. The diet is made as nutritious as possible. Unless there is diarrhea or vomiting, the patients may be given solid food, even if there is fever. Meat, eggs, and milk should constitute the major part of the dietary. If there is vomiting or diarrhea they must be treated on general principles. Constipation should be treated by enemata rather than purgatives. Flatulence and indigestion must be treated by regulation of the diet and the use of bismuth or soda. For the relief of pain the application of heat by the hot-water bottle or turpentine stupes may be employed. Occasional painting with iodine may also be effective. The ascites, unless excessive, may be let alone. If it seems necessary, the abdomen may be tapped, but not in the ordinary way. By reason of the adhesion of the viscera there is too great danger of wounding the intestine if a sharp trocar is plunged in, as is usually done. An incision should be made through the abdominal wall and a blunt trocar introduced. If this measure fails, laparotomy must be resorted to. Tonics, such as cod-liver oil, arsenic, or the syrup of the iodide of iron, are to be given when the condition of the stomach permits. In any case abundance of fresh air and attention to all the details of hygiene are of prime importance.

**Surgical Treatment.**—This consists in a free laparotomy with drainage of the peritoneal cavity. It is the only method advisable for cases of the ulcerative type or for any case in which the effusion is excessive, particularly if the effusion be purulent. It is advocated by some surgeons for all cases. The results of surgical treatment have, for the most part, been very gratifying. Rotch, of Boston, has reported 62 cases of tuberculous peritonitis observed by him, 32 of which were operated upon with 12 deaths; of the 30 cases not operated upon, 20 died. Aldiber reports 52 operative cases. One of the acute miliary type died after operation. Of 6 cases of subacute type with ascites, but one died, 8



per cent. recovering. Of 16 cases of chronic type with ascites, but one died, 93.8 per cent. recovering. Of 9 cases with encysted collections of fluid, all recovered. Of 6 cases of the fibroadhesive type, all recovered; and of 8 cases with suppuration, 7 recovered, but none of the cases had been followed more than a year. These very favorable figures have not been borne out by later reports, but the results have been such as to make operation advisable in any case not yielding promptly to hygienic and palliative treatment.

### TUBERCULOUS MENINGITIS.

By D. J. MCCARTHY, M.D.

Tuberculous meningitis may either be a local process affecting the meninges secondary to a tuberculous focus elsewhere, or it may be part of a general blood infection by the tubercle bacillus.

**Etiology.**—While the disease may occur at any time of life, it is much more frequent in childhood. It is rare during the first and second years of life, although it is quite possible that many cases of basic meningitis in infancy may belong to this affection. The largest number of cases occur between the second and fifteenth years of life. Operative procedures on tuberculous lymph nodes, adenoid growths, and especially on tuberculous joints, may determine a blood infection in which the brain and spinal cord are most intensely affected and dominate the clinical picture. In those cases in which the inflammation is a local process (not a manifestation of a blood infection) it is usually secondary to a tuberculous focus in the immediate neighborhood of the meninges, such as in the bones of the skull or of the spine, the cavities of the face, nasal fossæ, the orbit, or the ear. From these areas the infection may be direct by extension, but more frequently by lymphatic transmission. The source of infection, however, in the vast majority of cases must be sought for at some distant point, and is usually found in a local process in the lungs or the peribronchial lymph nodes. The affection may originate in tuberculosis of the abdominal organs, the joints, the bones, superficial lymph nodes, and even the skin. While the transmission in these cases is probably through the blood and the localization of the process in the meninges is due to the lessened resistance of these membranes to infection and the greater resistance of the other tissues of the body, it is, however, quite possible that lymphatic transmission may be of much more importance than has heretofore been suspected.

In that small group of cases in which a careful search at autopsy does not reveal tuberculosis elsewhere in the body the source of infection may be direct from the nasal cavities. The relation of adenoid growths in the nasal pharynx to affection of the meninges should be borne in mind. George B. Wood has recently called attention to the frequency of affections of the tonsils and adenoids caused by the tubercle bacillus.

**Pathology.**—The gross appearance of the brain of patients dying from tuberculous meningitis varies greatly. In older children and in adults there may be little evidence of a marked inflammatory process. Numerous small grayish granules are found on the outer surface of the bloodvessels and may be found only after careful searching. The pia may be apparently normal, slightly reddened at the seat of some of the small grayish granules. In infancy and early childhood the inflammatory process is much more distinct and intense than in adults. The inflammatory exudate at times has a semipurulent appearance, is present over the entire base of the brain, and extends along the bloodvessels toward the convexity or into the brain substance, with the production of local areas of inflammation sometimes of a hemorrhagic type. I have seen this exudate over an eighth of an inch in thickness around the optic commissure. The tubercle bacillus may be found in the granules, in the exudate, and the inflammatory areas in the brain substance. The examination of the insides of the bloodvessels may show an intimal tuberculosis (Heektoen).

Distention of the ventricles due to an internal hydrocephalus is present even in those cases in which there is an absence of exudate blocking up the communication of the ventricles with the subarachnoid spaces. This is probably due to a toxic irritation or actual inflammation of the ependymal lining of the ventricles. The brain substance is edematous and there is evidence in the flattened convolutions of intense intracranial pressure. In a small number of cases the spinal meninges may also be infected by the inflammatory process. While the process is usually confined to the cervical region, the entire cord is at times affected.

**Symptomatology.**—The symptomatology is so complex, varying with the intensity of the inflammatory process, the presence or absence of exudate, and the complicating cerebral intoxication that we shall divide the course of the disease into three stages—the stage of invasion, the stage of irritation, the stage of coma and paralysis.

**Stage of Invasion.**—For a varying length of time (one to several weeks) the child loses weight, is peevish, irritable, restless at night, grinds the teeth, has no desire to play, and is drowsy in the daytime. A slight temperature develops slowly, but usually does not rise above 100° or at the most 101° F. at night. The bowels become constipated although in a small number of cases there may be diarrhea. Headaches now develop and may be associated with vomiting. Headache may be present from the beginning of the prodromal period. The presence of headache, constipation, irritability, and vomiting in a child exposed to a tuberculous infection should put the physician on his guard for an oncoming meningitis.

**Stage of Inflammatory Irritation.**—As the inflammatory process becomes marked the fever increases somewhat, the child is evidently much weaker and very sick, and complains of light and sounds. The irritability is increased; the child lies in a semisomnolent condition, answering when spoken to in a peevish manner. Constipation is

marked; the pulse is slow and irritable. If the child is excited or disturbed by moving, the pulse will ascend from 60, 70, or 80 up to 140. The pulse may, however, be very variable. Other vasomotor phenomena are marked, such as alternate flushing and paling of the cheek and of the trunk. At times brilliant-red patches of irregular outline develop and last for several hours on one portion of the body and then disappear, reappearing later in other areas. In the later stages of the disease a distinct marbling of the skin may appear. The headache is more intense and the irritability is increased by a hypersensitiveness to touch of the entire body. Motor phenomena are usually very marked. Rigidity of the muscles of the neck and of the back, retraction of the head, grinding of the jaws, pulling up of the angles of the mouth, and strabismus are present. The rigidity may be so marked that the body may be lifted as one piece by elevating pressure on the occiput. The limbs may become rigid and contracted with increase of the reflexes. General convulsions may occur or may be absent throughout the entire course of the disease. Even in such cases there are twitchings and jerkings of the extremities. Partial convulsions may occur. At this stage Kernig's symptom is well developed. The child after several days becomes stuporous and may at times mutter to itself, but distinct delirium is comparatively rare. Variations in the respiratory rhythm are present throughout the entire stage and are almost characteristic. Even when the child is quiet there is a distinct irregularity of the rhythm, with inequality of the amplitude of the respiratory excursions, "a disharmony of association between the movements of the diaphragm and those of the thoracic walls." As the disease advances respirations become more irregular, a period of suspension of the respiratory movements being followed by long, deep, sighing respirations. Toward the end the respirations may follow the Cheyne-Stokes type.

The pupils early in this stage may be contracted, but later become dilated and at times may be unequal. The irritation of the oculomotor nerves at first produces a spasmodic internal strabismus followed by a paralytic squint. Slow movements of the eyes from one side to the other and even distinct nystagmus may be observed. The ophthalmoscope reveals in the majority of cases a moderate choking of the disk and in a smaller number of cases bright, shining spots on the choroid (miliary tubercles of the choroid), which when seen are absolutely diagnostic.

*Stage of Coma and Paralysis.*—The child is now unconscious; the spasm of the neck, of the back, and of the extremities relaxes; the pupils are markedly dilated, the eyeballs turned outward and upward, the lids are half-closed, and complete blindness is present. The pulse is very rapid, the respiratory rhythm is irregular, the superficial temperature is subnormal, the rectal temperature usually high, although it may be subnormal. Convulsions may occur, but are, as a rule, very light and limited as to time and distribution. They may, however, be as intense at the end as at the beginning of the second stage, and may be followed by a temporary paralysis. The paralysis of this stage is,



however, usually permanent, due to destruction of the nerve tissues. The extremities are flaccid and relaxed, there is complete paralysis of the eye muscles, a dropping of the angles of the mouth with loss of expression, and a paralytic condition of the jaw. Sometimes retention of urine occurs toward the end of this stage. As the end approaches cyanosis and lividity of the skin and mucous membranes appear, the extremities and trunk become cold, and death slowly takes place. Death sometimes follows a general convulsion.

The course of the disease varies with the intensity of the infection and of the inflammatory process and the age of the child. In infants a fatal termination may be expected within a week. Death may occur in children under two years of age in two or three days from involvement of the base and convexity. The onset is sudden, with headache, high fever, convulsions, and a rapid fatal termination before coma appears. In later childhood the disease runs a course of from one to two weeks. There are other cases running a subacute course and lasting from four to six weeks.

**Diagnosis.**—The diagnosis of tuberculous meningitis from other forms of meningitis depends on the discovery of some active or latent focus of tuberculosis elsewhere in the body and the presence of tubercle bacilli in the cerebrospinal fluid. While the clinical picture in some cases is typical it often does not differ essentially from that presented in other forms of meningitis. A prolonged prodromal period with constipation, bradycardia, slight elevation of temperature, with the pulmonary and ocular symptoms above described will differentiate the tuberculous from other forms of meningitis. The non-tuberculous forms of meningitis may be distinguished by the suddenness of onset, the absence of prodromes, the initial fever, and the rapidity of course. When the meningeal infection is a part of a general miliary tuberculosis affecting other organs a typhoid state may be presented, leading to a diagnosis of typhoid fever with symptoms of meningeal irritation. The absence of leukocytosis and the presence of the Widal reaction in the blood and the absence of tubercle bacilli in the cerebrospinal fluid should easily differentiate the two conditions.

From brain tumor in children the diagnosis is made by the slow onset of the symptoms in tumors with the absence of fever, the greater intensity of the optic neuritis, and the presence of localizing symptoms.

**Lumbar Puncture.**—The examination of the cerebrospinal fluid as a method of diagnosis, in tuberculous as in other forms of meningitis, is of great value. Puncture with a large hypodermic needle or small antitoxin syringe may be made below the termination of the spinal cord at the second lumbar vertebra without injury. Local anesthesia may be used and it is sometimes advisable to produce slight chloroform anesthesia. This is, however, in the great majority of cases unnecessary. Thorough cleanliness both as to instruments and the skin is essential to prevent infection of the spinal meninges. The point of the needle should be inserted between the spinous processes a little to one side of the median line. At a varying distance depending on



the age of the child (2½ cm. in infants) and the interspace selected, the needle will penetrate the spinal canal. The fluid runs drop by drop and something may be learned from its character. In tuberculous meningitis it is usually clear, and is as opalescent as the normal fluid; it may show a sediment on standing, or it may be turbid. In simple meningitis it may be clear. In purulent meningitis it is cloudy or distinctly pussy. Cover-slip preparations should be made and carefully studied both for the organisms and also for the character of the formed cellular elements. The technique for the examination of the fluid for the presence of tubercle bacilli is given by Hand<sup>1</sup> as follows: The fluid should be allowed to drop from the needle into a sterile test-tube, which is then stoppered with cotton and allowed to stand for several hours, or until a strand of fibrin has formed; this occurs in from one to six hours, and it either settles to the bottom or reaches from the top of the fluid down to the bottom, spreading out in a fan-shaped, delicate film. A straight platinum needle, not a loop, is touched to one edge of the fibrin, the adhesion being very firm; the fibrin is then transferred to a slide, care being taken to tip the test-tube so that the fibrin constantly floats in liquid; a few drops of the fluid are to be poured with the fibrin on to the slide, for, if the fibrin emerges for but an instant from the fluid, it will either roll up into a cord through which nothing can be seen or it will wrap itself so tightly around the platinum needle that it cannot be detached; to prevent this the edge of the test-tube should be flanged and not straight; when once on the slide and floating in the fluid, it can be carefully separated from the tip of the platinum needle with the help of an ordinary needle or pin; the excess of fluid is drained off from the slide and the remainder is evaporated by gentle heat, it being not only unnecessary but usually fatal to the success of the examination to press the fibrin between two slides; the film is fixed by heat, stained in the usual manner and then carefully gone over with a mechanical stage. A point for the protection of the examiner is worth mentioning; all of the germs are not caught in the fibrin, but some float free in the fluid, and as it is well to flood the slide even to the risk of overflowing, a blotter or piece of filter-paper placed beneath the slide will absorb both the fluid and the stray germs, and disinfection is then easily accomplished by combustion; if the blotter is dark in color, the film of fibrin can then be seen much more easily and located on the slide. The next step in the examination is the taking of cultures. After the chemical examination, for which 5 c.c. will suffice, the remainder can be used for inoculation into guinea-pigs if this is deemed advisable; this is hardly necessary if tubercle bacilli have been found, but it is very desirable in all other cases and should be carried out, if possible, for then the exclusion of tuberculosis rests on unassailable ground. The non-tuberculous cases do not show the fibrin formation in anything like the degree that tuberculous cases do; in the former there usually being a scanty, yellowish-white sediment

<sup>1</sup> Philadelphia Medical Journal, August 30, 1902.

of leukocytes at the bottom of the test-tube, extending for a short distance up the sides.

If this technique be carefully followed tubercle bacilli will be found in practically 100 per cent. of cases of tuberculous meningitis.

The chemical examination of the residual fluid may be made, and while it gives valuable data, is not as important as the microscopic examination. The changes to be expected are a diminution or absence of the normal sugar-reacting substance of the fluid, an increase of the albumin, and the presence of leukocytes. The normal quantity of copper-reducing substance in the fluid (sugar?) is 3 to 5 cgm. in 100. The normal amount of albumin is 0.25 part in 1000. A study of the cell elements in the fluid shows a variation in the different forms of meningitis. The polynuclear leukocytes are in the majority in the non-tuberculous forms of meningitis, the lymphocytes in the tuberculous form. In the epidemic form the diplococcus intracellularis will be found after properly staining cover-slips made from the sediment of the centrifugated fluid. Staphylococci, pneumococci, and other pyogenic organisms have been found in other forms of meningitis.

**Prognosis.**—Tuberculous meningitis may be considered to be universally fatal. In an extensive experience in the examination of the brain of adults and children dying from pulmonary tuberculosis at the Philadelphia Hospital and the Henry Phipps Institute I have seen cases which presented at autopsy evidence of healed tuberculous inflammatory lesions of the meninges. I have, however, never seen a case of tuberculous meningitis recover. Ord and Waterhouse report a case of recovery by trephining and draining. I have seen only one case treated in this manner with an unfavorable result. Furbringer reports a case of recovery after spinal puncture, tubercle bacilli being found in the cerebrospinal fluid. The record of two cured cases in the literature calls attention to the hopeless nature of the affection.

**Treatment.**—The treatment is entirely symptomatic. A purgative in the early stage is indicated; ice-bags to the head or along the spine to control the pain, a proper nourishing diet, and a quiet, darkened room will add to the comfort of the patient. The surgical treatment (opening the skull by trephining, and, more recently, by a large osteoplastic flap practised by Agnew in 1891, and by Ord and Waterhouse, Jaboulay and others has been successful only on the one case above referred to, but in view of the hopeless nature of the affection it is deserving of more extended practice. The theory upon which this treatment is based is, that the exposure of the meninges to the air should have the same beneficial effect as in tuberculous peritonitis, and should also relieve the increased intracranial pressure. The use of lumbar puncture in cases where the cerebrospinal communication is open produces an amelioration of the symptoms without, however, any permanent results.

## CHAPTER XVI.

### DIPHTHERIA.

By MATTHIAS NICOLL, Jr., M.D.

**DIPHTHERIA** (Greek, *διφθερία*, a skin or membrane) is an acute infectious disease, due to the presence and growth of the Klebs-Loeffler bacillus on a mucous membrane or wound of the skin surface, upon which it produces a pseudomembrane. General symptoms especially referable to the nervous system are caused by the elaboration of certain toxins chiefly at the point of inoculation.

The pathological changes in the organs are caused in great part by toxemia, but also by the presence and growth of the Klebs-Loeffler bacillus alone or in combination with other organisms within the tissues.

No part of the earth seems to be free from at least occasional outbreaks of the disease. In large cities it is endemic, the cases varying in different seasons and years, in number and average degree of virulence. In country places and small towns it occurs as a local epidemic, one or more cases being brought into a community from extraneous sources and spreading the disease.

From an analysis of a large number of cases in this country, Continental Europe and England, one may conclude that the disease is considerably less frequent during the warmer months of the year, and this is readily explained by the fact that during these months the people live more out-of-doors, the children leave their overheated and overcrowded houses for a greater part of the day, schools are closed, and the prevalence of catarrhal affections of the upper air passages is greatly diminished.

**Etiology. Modes of Infection.**—Notwithstanding the impossibility of tracing the source of many apparently isolated and puzzling cases of diphtheria, it may be stated positively that one case of the disease always arises directly or indirectly from another. The most frequent methods of infection are by the inspiration of air, especially in closed rooms infected by a diphtheria patient, the use of handkerchiefs and towels in common, from handling infected toys, books, and clothing, and later by transferring the fingers to the mouth, the use of infected spoons, dishes, and food, and by kissing upon the mouth.

Physicians and nurses who do not take proper precautions in disinfecting their persons and clothing are frequently the means of carrying contagion from infected to healthy individuals.

The theory of indirect infection presupposes what we know to be a fact, that the diphtheria bacillus may live for weeks and months, not



only in throats which have every appearance of health, but also dried upon clothing, bedding, wall-paper, carpets, etc., which have not been properly disinfected. An unusual but well-authenticated method of transmission is by means of a milk supply contaminated by dairy helpers who are afflicted with the disease. Milk is an excellent culture medium for the Klebs-Loeffler bacillus and thus affords a ready method of conveying actively growing colonies to consumers of the infected supply.

Transmission of diphtheria by domestic animals has not been substantiated, nor its conveyance by means of defective drainage and sewer gas proved. Nevertheless, unsanitary conditions of drainage tend to prolong the presence of the diphtheria germ when once implanted.

*Predisposing Factors.*—No race can be said to be immune to the disease. According to some observers, negroes show a greater degree of resistance than the white races. By some, the Jews are thought to be especially susceptible, but this apparent susceptibility may readily be accounted for by the fact that the poorer Jewish quarters are usually those in which overcrowding and lack of sanitary precautions are most in evidence.

Age has an important bearing on its occurrence. Children under one year of age and especially those in the first six months of life possess a relative immunity. The ages from three to five years may be set down roughly as the time of greatest susceptibility; from the ninth to the tenth year the susceptibility slowly decreases, and from this period rapidly decreases.

The disease affects both sexes in about equal proportions. As with other acute infections, diphtheria attacks by preference those of low vitality and especially the subjects of chronic catarrhal conditions of the upper air passages and hypertrophy of the neighboring lymphatic structures (tonsils and adenoids).

**Bacteriology.**—The bacillus described by Klebs and Loeffler in 1883-1884 and later shown by Roux and Yersin to be the cause of diphtheria has been exhaustively studied. It is capable of exhibiting quite a wide degree of structural difference, even in the same culture medium, depending on the length of time a culture is grown, the consistence of the medium, temperature, etc.

Grown on Klebs-Loeffler serum, the medium most generally used for diagnostic purposes, for twelve hours or more at a temperature somewhat below 100° F., and stained with alkaline methyl-blue solution, the bacilli are seen as fine rods, straight or slightly curved, usually noticeably clubbed at one or both ends and arranged in larger or smaller groups with great irregularity; occasionally end to end in a broken line, but more often one bacillus forming an angle with another, a parallel arrangement not being commonly observed. The length varies from 1 to 6 $\mu$ , the width from 0.3 to 0.8 $\mu$ .

Common variations from the above are bacilli pointed at one or both ends, thick at one end and pointed at the other (so-called wedge shaped). Thick and short forms are occasionally met with resembling so closely



certain of the pseudodiphtheria bacilli that the true nature of the organism can only be positively determined by clinical symptoms, culture methods, and animal inoculation.

Neisser's stain is often used to bring out certain morphological characteristics more clearly than can be done with the Klebs-Loeffler stain. It is made as follows: Solution No. 1: 1 c.c. methyl blue dissolved in 20 c.c. 96 per cent. alcohol, 90 c.c. distilled water, 50 c.c. glacial acetic acid. Solution No. 2: 2 c.c. vesuvin to 1 litre of boiling distilled water. Stain in No. 1 for three to ten seconds, stain, wash in water, and stain in No. 2 for three to five seconds. Wash off and examine.

The body of the bacillus will thus be stained a brownish color, while the so-called polar granules of Neisser-Ernst will be seen at one or both ends of the rod as dark-blue oval bodies, the diameters of which are invariably somewhat greater than that of the bacillus.

The chief characteristics of the Klebs-Loeffler bacillus may be set down in brief as follows: It is non-motile, and while growing more luxuriantly in the presence of oxygen, thrives also without it. It does not form spores, but will live when dried for weeks and months, especially when protected from sunlight. It is readily killed by a temperature of 136° F. It is not killed by freezing temperatures, beginning to grow at a temperature of 20° C. (68° F.), but most luxuriantly about body temperature (96° to 99° F.).

On apparently healthy mucous membrane the bacillus may exist for months, both in those who are convalescing from the disease and in the throats of those who have never exhibited any symptoms of it, but who consciously or unconsciously have been exposed to infection. Such bacilli, while not apparently harmful to those who must in consequence be regarded as possessing a natural or acquired immunity, are nevertheless sources of great danger when transferred to susceptible individuals, and it is probable that recurrent attacks of the disease are often due to the presence of these latent germs, which take on active growth, by reason of a discontinued immunity, from temporary, general, or local pathological conditions.

On blood serum after twelve hours' growth the colonies of Klebs-Loeffler bacilli are seen as milky-white, gray, or yellowish points, slightly elevated with irregular borders. Neighboring colonies may coalesce. The serum is not liquefied.

In bouillon (alkaline, slightly acid or neutral) the bacilli grow readily, producing acid in their growth. Of other media, milk may be mentioned as a favorable one, its appearance not changing through the growth of the organism.

According to Dr. W. H. Park the bacillus is pathogenic for guinea-pigs, rabbits, chickens, birds, and cats. Moderately so for dogs, goats, cattle and horses, and not for rats and mice.

Diphtheria bacilli differ widely in their virulence, from those which produce death with fearful rapidity to those which, apparently possessing all the cultural and morphological characteristics of the former, are absolutely non-virulent.

Between these two classes may be mentioned a type of bacilli which when inoculated into guinea-pigs produces a chronic disease, slow in its course and ending fatally by inducing a state of general inanition.

It has been shown that the virulence of certain avirulent bacilli may be restored to them by passage through the bodies of animals.

The characteristic lesions produced by inoculating animals with diphtheria are identical with those found at autopsy on human beings dead of the disease.

For a long time it was supposed that the bacillus of diphtheria, when implanted upon a mucous membrane, showed no tendency to invade other structures save those in direct continuity with the site of the lesion, trachea, lungs, etc. Careful investigation of the various organs, however, show this not to be the case, but that the bacillus may be carried by the blood and lymph stream to all parts of the body. It is found in pure culture, or associated with other organisms—namely, streptococci, pneumococci, and staphylococci. This association is especially seen in the lungs. In the other organs, liver, spleen, etc., the bacilli may be found alone.

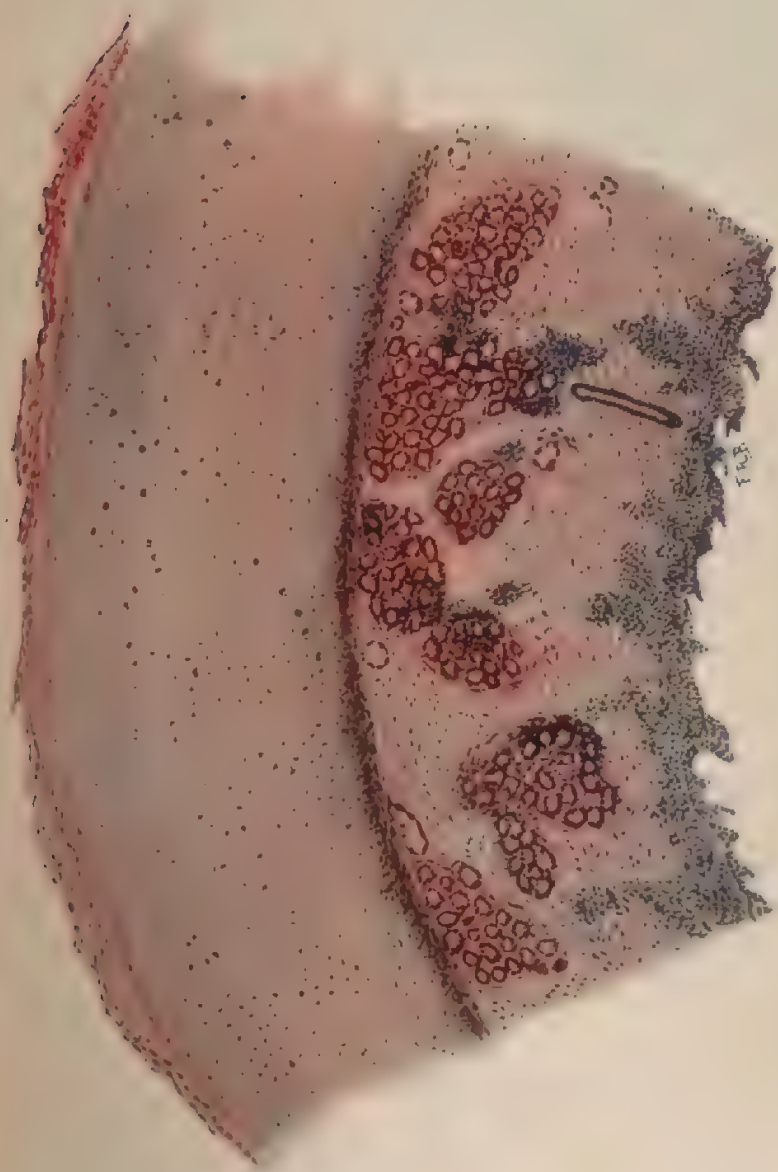
How great a part this migratory bacillus takes in producing the symptom-complex of the disease, as well as the local lesion found in the organs, cannot be definitely determined. As a rule, the migration seen in severe and especially in septic cases, although not confined to the latter. It is reasonable to suppose that wherever in the body living virulent diphtheria bacilli are found, that they perform their share in producing the toxemia peculiar to the disease.

In the lungs, the accessory sinuses of the nose, and middle ear, the association with other organisms produces marked pathological changes.

The most frequent and dreaded complication of the disease, bronchopneumonia, is due not only to the action of the toxins on the pulmonary structure, but to the actual presence of the bacillus within them, always associated with other organisms.

The question as to the relation between certain diphtheria-like bacilli (pseudodiphtheria bacilli) and the bacillus of Klebs-Loeffler cannot here be entered into at length. Sufficient to say that a minority of observers regard the former as but varieties of the true organisms, while the majority hold the opposite opinion—namely, that the Klebs-Loeffler bacillus, while it may be absolutely avirulent and lacking in certain cultural and morphological characteristics, belongs to a distinct class, and under no circumstances whatever can the pseudobacillus be made to possess all the characteristics of the Klebs-Loeffler bacillus.

It is at times very difficult, even impossible, to state positively that such and such an organism belongs to the class of Klebs-Loeffler bacilli, judging from a morphological standpoint, especially in the absence of clinical data. Fortunately this is not a very common occurrence, and repeated cultures will generally serve to settle the question of diagnosis.



Section through a Diphtheria Membrane of the Larynx.





such doubtful bacilli are more often found in cultures from the nose and conjunctiva than in those from the throat.

The most distinctive characteristics of the pseudobacilli may be summarized as follows: They are apt to be thicker and shorter than true bacilli, are often arranged in parallel groups; when stained with Leisser's solution they show no or only atypical polar granules, they do not produce acid in bouillon, and are not pathogenic for guinea-pigs.

To such rules, however, there are many exceptions, and, as already stated, bacilli are occasionally found which fulfil all or nearly all these conditions, and yet must be classed as true diphtheria.

The more difficult cases fall naturally to the expert bacteriologist, in the absence of whom the practitioner must rely on the clinical symptoms taken in conjunction with the morphological characteristics to establish a diagnosis.

**Pathology.**—The pseudomembrane may be situated on any mucous surface, but most frequently on those contiguous to cutaneous areas. If the latter are denuded of epithelium they also form suitable sites for the propagation of disease.

**The Pseudomembrane.**—The faucial tonsil is the most common site of the membrane, together with the adjacent parts of the pharynx. Next, and with about equal frequency, the nasopharynx, and lastly, the larynx and trachea. In the severe so-called toxic forms of the disease it often spreads to all of these areas. It is gray white, yellow, less often dark in color. It may be very thick or practically invisible; clings closely to the underlying surface, or be easily removed in large flakes; the latter is characteristic of laryngeal and tracheal pseudomembranes.

The process by which it is formed is that known as coagulation necrosis, the necrosis, as a rule, involving only the superficial underlying structures. More rarely there may be deep destruction of tissue, a process more often seen when other organisms are associated with the Klebs-Loeffler bacillus. (See Plate IX.)

On microscopic examination of a section of tissue, underlying a diphtheria membrane, it is seen that the epithelium beneath the latter is destroyed to a great extent; there is an extensive leukocyte infiltration of the tissues, extending to a variable depth beneath the surface, together with granular particles, remains of cell nuclei, and a greater or less number of red blood cells. Beneath the area of cell infiltration the tissues are filled with fibrinous exudate and red blood cells.

The change in the bloodvessels consists of thickening of the walls and plugging of their lumen with fibrinous masses. The mucous glands may show a mild form of acute degeneration or complete necrosis of their structure.

The processes described are usually limited by the membrana propria, but in some cases this boundary is crossed, and the tissues beneath it are invaded by fibrin and cell infiltration.

The one characteristic pathological change caused by the toxins of diphtheria is that which involves the nerve structure. This consists of

parenchymatous and interstitial degeneration of the peripheral nerves, and in all probability of certain degenerative changes in the spinal cord. Other conditions that have been described are hyperemia, hemorrhage, and fatty degeneration. The cases in which the nerves are affected are usually those of long duration, or those in which there is extensive membrane production and consequent marked toxemia.

In the heart there may be cell infiltration of the myocardium, fatty infiltration and degeneration, or interstitial changes with fragmentation of the muscle fibres.

Pulmonary lesions should be considered rather as a complication than as a part of the disease, for it may be concluded, from the results of experiments and postmortem bacteriological findings, that in the production of these lesions the Klebs-Loeffler bacillus plays only a preliminary part, the real lesion being the work of associated organisms, notably the streptococcus; less often the pneumococcus.

The lesions of the lymphatic structures, spleen, lymph nodes, etc., consist in brief of cell hyperplasia, general congestion and areas of cell necrosis, so-called focal necrosis. The latter is not peculiar to diphtheria, but may occur in all severe acute infectious diseases if sufficiently prolonged. Hemorrhages are frequently seen.

The diphtheritic membrane may invade the alimentary canal in any part of its course, as an extension of the disease from above downward. Diphtheria of the stomach is not infrequently found at autopsies.

The lesions of the liver consist of small areas of necrosis resembling to the naked eye miliary tubercles and due to the action of the specific toxin on such areas of liver cells as are supplied by bloodvessels whose walls have been affected by the disease.

In the kidneys there are no lesions characteristic of the disease. The one most commonly found is that of acute degeneration to a greater or less extent. Acute interstitial lesions occur rather infrequently.

The voluntary muscles show similar changes to those described as occurring in the myocardium.

**Symptomatology.**—Many different classifications of diphtheria have been attempted, none of which is entirely satisfactory. The purely bacteriological classification, while having a scientific basis as a recommendation, leaves out of account the variable reaction of different constitutions to the same germ or combinations of germs. On the other hand, a classification based on purely clinical observation is illogical, since only knowledge of the bacteriological findings in many cases will enable us to understand why those, seeming to all appearances identical, show such a variation in clinical symptoms.

For practical purposes the classification based on the location of the membrane and the character of the organism or combination of organisms which take part in its formation is perhaps the most satisfactory.

Pure or *fibrinous* diphtheria is due to the action of the Klebs-Loeffler bacillus alone, the severity of the cases depending on the extent of

the membrane and its location and the degree of resistance shown by the individual to the action of the toxin.

This form of diphtheria is less frequently followed by complications, yields more readily to specific treatment, and is somewhat more frequently seen amid more favorable surroundings than in institutions and tenements.

*Mixed* diphtheria is due to the association of the Klebs-Loeffler bacillus with other organisms, usually the streptococcus. It is usually characterized by its greater severity, the tendency to complications, resistance to antitoxin, and its proneness to attack those in previously poor health, especially the subjects of enlarged lymph nodes. It is the usual form which complicates scarlet fever and measles and is the form occurring as a primary disease of the nose.

The name *catarrhal* diphtheria has been given to that form of the disease in which there is no visible membrane. It is not of common occurrence and derives its importance not so much from the danger to the patient as the probability of its being transmitted to others in a less benign form, especially as the diagnosis is not usually made except when the nature of the disease is suspected, as after the exposure of the patient to a known case of diphtheria, when the bacteriological examination serves to clear up the nature of the case, the symptoms being identical with ordinary catarrhal pharyngitis.

**General Symptoms.**—The temperature curve of uncomplicated diphtheria follows no particular course. In the pure form of the disease the fever is not apt to be high at any time. Indeed, in older children unless a careful record be kept, there may appear to be little or no fever, in this differing from the follicular tonsillitis. The fever increases with the formation and spread of the membrane, and steadily declines with its disappearance. In younger children the temperature is high for a day or two, after which it slowly declines. With the appearance of various complications there is a rise of temperature, especially with involvement of the lungs. The action of the toxin is invariably shown by an increase in the pulse rate, although the pulse may be slow at the onset. In very young children it is especially affected. In older children a continuously high pulse, 150 or more to the minute, may be regarded as a complication. Other conditions of the pulse which probably justify the suspicion of myocardial changes are bradycardia, irregularity, and a weak, thready action.

**Blood.**—The toxins of diphtheria produce certain changes in the blood, the most constant of which is a leukocytosis, varying in degree with the extent of the membrane, the virulence of the individual organism, and the amount of reaction on the part of the patient. The condition begins with the disease and reaches its height at the height of the latter, and then gradually declines. It is prolonged by the occurrence of various complications, especially bronchopneumonia. The polynuclear elements are those most affected. The increase of these may be very marked, especially in cases which terminate fatally.

The red cells are diminished to some extent, and also the hemoglobin,



and, according to some observers, the specific gravity of the blood is increased together with its coagulability.

*Urine.*—Apart from the various forms of nephritis which occur as a complication of diphtheria, certain changes in the urine are commonly observed even in mild cases as a result of toxemia. The quantity may be diminished to a greater or less extent, occasionally suppressed. Albumin occurs in one-third to one-half of the cases during the course of the disease. It is usually small in amount, occasionally accompanied by casts and is due to degeneration of the renal epithelium. It usually clears up shortly after the disappearance of the membrane. The severe forms of nephritis are not commonly seen in uncomplicated cases. In the mixed cases and especially when the disease complicates scarlet fever, nephritis is very common.

*Diphtheria of the Tonsils and Pharynx.*—This most common form of the disease varies in its clinical course from an attack so mild that the children do not seem to be at all ill and only an examination of the throat reveals the true nature of the case, to that with a rapidly spreading membrane which includes the whole throat, and if not treated at the very onset causes death by an overwhelming toxemia.

In the mild form there is seen on one or both tonsils a white or gray patch which either covers the tonsil or resembles a punched-out area with membrane at the bottom. The tonsil is swollen and reddened; the membrane is usually friable and may be removed in small pieces, leaving a bleeding surface. At times the tonsils present the appearance of an acute follicular tonsillitis, so much so that cultures alone will serve to distinguish the two diseases. The membrane may remain confined to the tonsil or spread to the posterior pillar of the fauces, one or both sides of the uvula, and back of the pharynx. Rarely it is entirely invisible by the ordinary methods of throat examination, being concealed behind a swollen tonsil, or is back of the uvula and soft palate.

I have recently seen a case in which the throat presented every appearance of a scarlatinal angina, both tonsils being bright red and swollen, together with the fauces; an antitoxin erythema added to the difficulties of the diagnosis. On pulling one tonsil slightly forward a large membranous patch was seen on its posterior aspect, the culture showing Klebs-Loeffler bacilli.

The symptoms in mild cases are not marked. The child, if old enough, complains of sore throat and some difficulty and pain in swallowing. The pulse rate is increased, and there are two or three degrees of fever. Provided the membrane shows no tendency to spread, it begins to disintegrate after a few days to a week. Paralysis, usually confined to the pharynx, occasionally follows. A moderate albuminuria is regularly present. The cervical lymph nodes are somewhat swollen and tender (Fig. 78).

In the severe form of the disease the picture is one of overwhelming toxemia. The membrane may not differ in appearance from that of the benign form, but, as a rule, is of a dirty brownish color, or yellowish



PLATE X



Diphtheria.



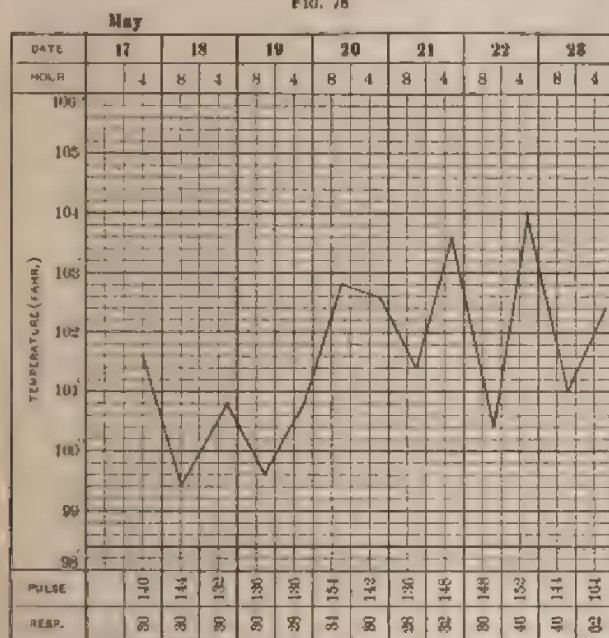
Pseudodiphtheria.



rather than white. The parts adjoining the tonsils are rapidly involved. The uvula, posterior pharynx, and fauces are covered by a practically continuous thick membrane, so that when one looks at the throat the separate structures may be almost unrecognizable. The tonsils are greatly swollen, and such parts of them as are not coated with membrane are a dusky or bright red. The uvula is often edematous. The process is very liable to extend upward to the nasopharynx. The cervical lymph nodes involved early in the disease are large and tender.

At the beginning of the attack there may be a chill or convulsion. There is often a low delirium, followed by somnolence; food is taken with great difficulty both on account of the narrowing of the passages

FIG. 7B



Temperature chart of case of diphtheria. Swelling of lymph nodes.

and the pain caused by swallowing. The pulse is rapid and small, sometimes irregular. The temperature is high.

In this form of pharyngeal diphtheria, as in the benign, cultures from the throat will show at least a predominance of the Klebs-Loeffler bacilli. Those taken from the nose usually show a greater number of cocci. When the latter predominate the type of the disease presents certain differences from the form just described. In the mixed form of the disease the patient suffers from the combined effect of the activity of more than one set of organisms. The membrane is apt to be discolored and it may be even black when there is much hemorrhage beneath it. It usually spreads rapidly to all adjoining surfaces. The parts are greatly swollen and there is a constant discharge from the nose and

throat of a thin fluid mixed with mucopus, blood, and pieces of membrane. The appearance of the patient is that of one suffering from general sepsis. The pulse is rapid, the temperature of a pyemic type, often very much elevated, showing marked and rapid remissions. The kidneys are affected early in the disease, and other complications are frequent.

*Nasal Diphtheria.*—The common form of this disease is seen in children past the age of infancy. It is of frequent occurrence in institutions for children and in schools, probably much more so than is generally supposed. When it is confined to the nasopharynx and anterior nares the children seem to be suffering from an aggravated rhinitis. There is a constant nasal discharge of a thin or mucopurulent character, often mingled with blood and causing excoriation of the nostrils and upper lip. There is more or less obstruction to nasal respiration and consequent mouth breathing. The patients do not seem particularly ill, as a rule merely uncomfortable. There may be a moderate toxemia, which is shown by lassitude, headache, anorexia, and slight fever. The nature of the disease is often no doubt overlooked, and can only be determined by a bacteriological examination, although occasionally a careful inspection of the anterior nares will reveal the presence of membrane, usually on the septum and deep in the canal, and the lymph nodes below the angle of the jaw will be found enlarged. The mucous membrane is reddened and swollen. The cultures usually show mixed infection. The process may extend to the nasopharynx and even to the larynx. These patients are a source of grave danger to those with whom they come in contact. Their handkerchiefs and fingers, constantly saturated with the infected discharges, are eminently suited to spread the disease.

When, instead of being confined to the anterior and posterior nares, these parts are secondarily infected from disease of the lower pharynx and tonsils, the symptoms are of great severity, especially as it is the type of disease seen most frequently in young children. If nurslings, they are unable to take nourishment, the mouth is held widely open, the respiration snuffling and snoring. The children are unable to rest, tossing restlessly from side to side. There is real obstructive dyspnea, the air not being able to enter the nose at all, and only insufficiently through the mouth. On inspiration there may be recession at the epigastrium, though usually not so marked as that seen in laryngeal diphtheria. There is marked toxemia. The children are pale, apathetic, with cyanotic lips and extremities; the pulse is rapid and feeble. The temperature is high. The lymph nodes of the neck are swollen. The children may die of toxemia, suffocation, or finally of extension of the disease to the larynx (Fig. 79).

*Laryngeal Diphtheria.*—The term croup may be applied correctly as a purely clinical description of an acute laryngeal obstruction, due to inflammation of the mucous membrane, together with spasm of the vocal cords. Membranous croup may be caused by the Klebs-Loeffler bacillus, either alone or in combination with other organisms, or ve-



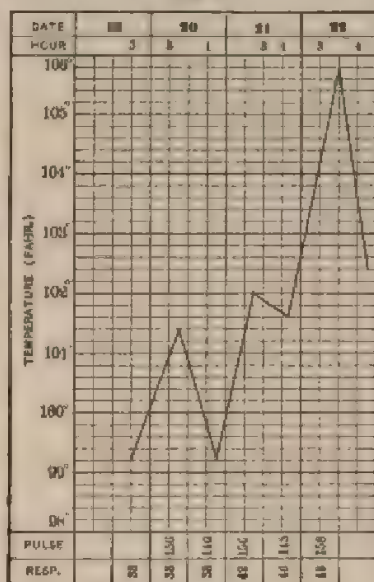
rarely by the streptococcus alone, and usually as a complication of one of the exanthemata. In order to avoid ambiguity it is perhaps better to employ the terms diphtheritic and non-diphtheritic for membranous croup, and limit the term catarrhal to that form of the disease which is due to simple acute catarrhal inflammation or congestion of the larynx.

Diphtheritic membranous croup is met with, as a rule, as an extension of the disease from the throat or nose. Less frequently the first symptoms noticed are those referable to the larynx, the throat only being slightly congested or absolutely normal. In the first instance the pharyngeal or nasal diphtheria may run for several days or even weeks before extending to the larynx, or it may do so in a few hours. Whether secondary or primary, the occurrence of laryngeal involvement produces a fairly definite train of symptoms only varying in the rapidity with which they follow one another or are modified by local or general treatment.

The course of laryngeal diphtheria may logically be divided into three fairly well-defined stages. In the first stage there is a hoarse, croupy cough, differing not at all from that so commonly observed in children at the onset of an attack of catarrhal laryngitis or tracheo-bronchitis. In the rare instances when a view has been had of the larynx at this stage there has been seen congestion of the mucous membrane and vocal cords. Cultures at this stage, unless special care be taken to introduce the swab actually within the larynx, often prove negative, even though later cultures show Klebs-Loeffler bacilli frequently in pure culture. The duration of this stage varies from a few hours to a day or two.

The second stage corresponds to the formation of the pseudomembrane within the larynx. The cough increases in frequency. It is brought on by disturbing the patient, by the taking of food or medicine, and by exposure to draughts and by crying. It is paroxysmal in character, and distinctly laryngeal, the patient acting as though particles of dust or other foreign substance were irritating the larynx. The cough is dry and ineffectual. During the attack the face becomes red or dusky, the bloodvessels become prominent, and the eyes bulge and lacrymate. There is soon developed partial or complete loss of voice; the respiration has a sibilant character as though a large volume of air was being

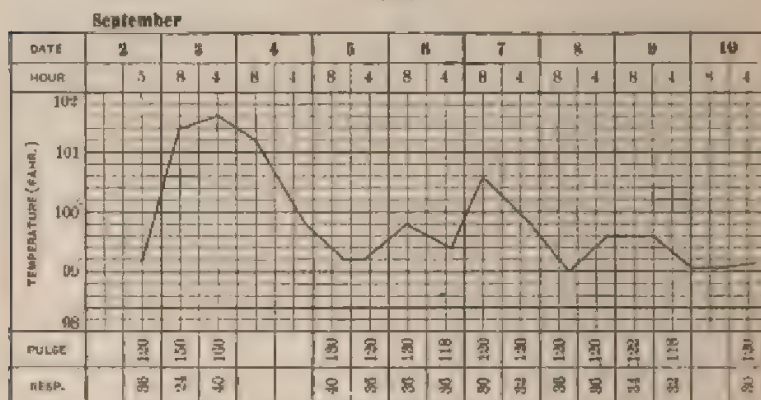
Fig. 79



Diphtheria with toxemia.

hurried through a small tube. The expiration is rude, and the pause between inspiration and expiration is marked. Even at this stage, if the children are kept perfectly quiet, their breathing is not so very labored during a great part of the time, but from time to time exacerbations of dyspnea occur, during which the children toss about, their face expressing great anxiety, the mouth partly open, chest heaving, the accessory muscles of respiration taking part in the process. At the height of inspiration there will be noted a marked recession of the soft parts at the epigastrium and above the clavicles. The respiratory murmur at the bases of the lung on auscultation will be found to be diminished. After the attack passes the child sinks back exhausted. It is repeated at shorter and shorter intervals. Occasionally the automatic expulsion of pseudomembrane terminates the attack (Fig. 80).

FIG. 80



Laryngeal diphtheria. Expulsion of membrane.

From postmortem examinations of the larynges of children who have died at this stage, it is seen that the amount of membrane within the larynx and involving the vocal cords can by no means be estimated from the character of the symptoms. With the severest variety of laryngeal dyspnea there may be only moderate superficial ulceration of the cords and little or no membrane external to them, the larynx being invariably congested. On the other hand, the membrane may be found to extend from the larynx in a continuous layer even beyond the bifurcation of the trachea. Such great extent of membrane, however, is to-day rarely seen, except in such cases as have not received antitoxin at all or too late in the disease to be effectual.

In the third stage dyspnea is marked and constant. The exacerbations and remissions of the previous stage are not seen. The respiration is carried on with great difficulty, all the accessory muscles being constantly called upon. The child sits up, or tosses from side to side. The recessions previously noted are more marked and constant. The inspiration and expiration are noisy and perfectly typical of the disease.

It is now evident that there is a continual lack of oxygen reaching the lungs. The lips and fingers are blue, the skin bathed in perspiration, the pulse rapid and feeble. If the condition be not relieved by surgical means, symptoms of carbonic acid poisoning soon develop and the patient lies quietly, almost lifeless, except when an attack of coughing arouses him to feebler and feebler efforts to overcome the obstruction. He becomes almost pulseless, the face dusky, the skin clammy, the stupor deepens into coma, and the patient ceases to breathe. A convulsion occasionally ends the scene.

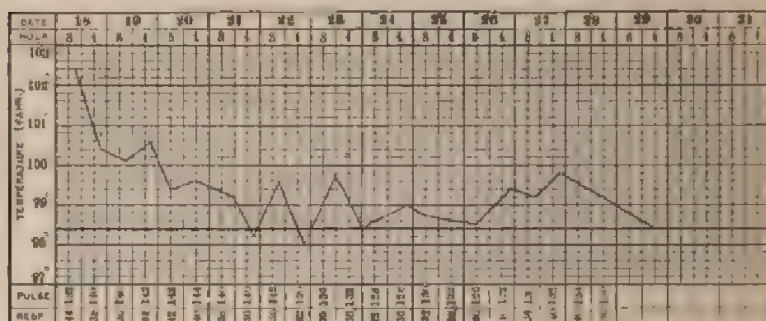
In explanation of the cause of laryngeal stenosis several theories have been offered. Those which have met with most general acceptance are briefly as follows: In the earlier stages of the disease, before the formation of pseudomembrane, a certain amount of obstruction is due to the swelling of the mucous membrane of the larynx and congestion of the vocal cords. The exacerbations of dyspnea are at this stage wholly due to spasm of the cords which in some way, not understood, have been rendered hypersensitive to stimulus. Such a condition may be seen also in whooping-cough. At a later stage, when the dyspnea is unremitting, it is due to the obstructing membrane and increased swelling of the parts, although the spasmodic element is still in evidence. In the final stage the latter does not come into play: the muscles as well as the skin of the body appear wholly irresponsive to stimulation, due, it is believed, to systemic carbonic acid poisoning, and the dyspnea is then wholly caused by the narrowing of the calibre of the laryngeal passage by congestion, pseudomembrane, and often by edema below the larynx. Upon this theory it is easy to explain the temporary relief afforded in the early stages by measures taken for the relief of spasm, such as the administration of emetics and sedatives, the applications of heat and steam, and the enforcement of perfect freedom from excitement of any kind; while the importance of the role played by the laryngeal membrane in the late stages of the disease is shown by the relief of all symptoms on the rare occasions when the membrane has been expelled by coughing.

*Diphtheria of the Trachea and Bronchi.* The laryngeal pseudomembrane may extend downward, involving the trachea, the larger and even the smallest bronchi. The name "ascending croup" has been given to a rare condition in which the diphtheritic membrane apparently first finds lodgement in the trachea or bronchi, and secondarily involves the larynx. Such a condition can only be positively diagnosed by the coughing up of a tracheal or bronchial cast with a relief of all the symptoms, but this state of affairs may be suspected when intubation or tracheotomy has failed to overcome the dyspnea, or occasionally by actually seeing the membrane through the tracheotomy wound. The only characteristic physical sign is that produced by occlusion of a large bronchus with consequent diminished or absence of breathing over that part of the pulmonary surface to which its ramifications lead. The symptoms are rapid breathing with real unremitting dyspnea, unrelieved by operation, and evidences of profound toxemia.



*Conjunctival Diphtheria.*—This occurs either as a primary disease or follows infection carried by the hand from the nose or throat. Three forms have been described. That most commonly seen is in part, at least, a true interstitial process and is probably always due to mixed infection. The lids are stiff and thickened, so that their eversion is difficult and frequently impossible without using a great deal of force. The conjunctiva of one or both lids is covered with a closely adherent blood-flecked membrane of a dirty-gray color, and there is a profuse purulent discharge. After the detachment of the membrane ulcerations, adhesions, and cicatrices may be left or the sight permanently destroyed (Fig. 81). In the second and less severe form the membrane is usually white, less adherent, the lids not thickened to any great extent, and, except for the presence of the membrane, the cases resemble those of acute catarrhal conjunctivitis. Complications are not common under proper treatment. In the third form there is no visible membrane, the conjunctiva is swollen, and there is a thin, glairy discharge, with no evidence of epithelial desquamation. This form of

FIG. 81



Conjunctival diphtheria.

the disease is probably not common and can only be diagnosticated by culture. Gonococci and staphylococci may be associated with the Klebs-Loeffler bacillus, but very frequently the latter is found in pure culture, except in the first form of the disease. The symptoms in general are as follows: The eyes are closed, the bulbs tender on pressure; when the lids are forced apart there is photophobia and epiphora, and the cheeks are often excoriated from the irritating discharge from between the lids. The constitutional symptoms consist of a moderate rise in temperature and pulse rate and other evidences of slight toxemia.

*Diphtheria of the Genitals.*—This invariably occurs as the result of autoinoculation from other sites. The disease is not a common one, and is seen most frequently in little girls. The membrane extends over both labia majora and minora, and occasionally to the vagina and anal margin. The parts are swollen, painful, and bleed easily. The inguinal lymph nodes are usually involved.

*Diphtheria of the Mouth.*—This is occasionally seen in severe cases of mixed infection occurring in the course of the exanthemata (scarlet



fever, measles, etc.). The patches are on the mucous membrane of the cheek, lips, and tongue, and involving fissures about the corners of the mouth and lips. The mucous membrane is reddened and bleeds easily. There is an increased flow of saliva and the breath has an odor similar to that in ulcerative stomatitis. The submaxillary lymph nodes are swollen, often to a great degree.

*Diphtheria of Wounds.*—This is seen, as already mentioned, as an extension of the disease from within the mouth to fissures about the lips. It also occurs in abrasions about the ear and nose, but may involve any cutaneous surface denuded of epithelium by scratching, eczema, herpes, etc.

*Diphtheria of the Ear.*—The Klebs-Löffler bacillus has been found in a large number of cases in the middle ear in cultures taken after death from diphtheria. It is usually associated with other organisms of the pyogenic variety and is merely an evidence of general infection, there being no true pseudomembrane present. In these cases the symptoms during life have been those of an ordinary suppurative otitis media.

A few cases of true diphtheritic infection have been described with membrane to be seen deep within the meatus after the rupture of the drum. There is a bloody, irritating discharge which excoriates the canal and external ear. As to whether this disease is carried by way of the Eustachian tube or the blood and lymph stream there is ground for a difference of opinion, as in some cases the former has appeared to be perfectly normal when diphtheria was found in the middle ear.

*Complicating Diphtheria.*—Measles, scarlet fever, and less frequently whooping-cough render a patient very susceptible to diphtheria infection, the natural protective barrier of the healthy mucous membrane being destroyed by the inflammatory processes accompanying the primary disease. The association of measles and scarlet fever with diphtheria is one much to be dreaded. The type of the disease is invariably that of a mixed infection, the streptococcus being responsible for the character of the local and systemic symptoms. In this type severe complications are the rule. There is usually great involvement of the lymph nodes, with suppuration and sloughing of the involved tissues. Gangrenous processes are occasionally seen, involving especially the maxillæ, adjoining soft tissues, and the ear. Bronchopneumonia is not uncommon, and also general septicemia.

*Complications and Sequelæ. Nervous System.*—Taking an average of a large number of cases, compiled by different observers, postdiphtheritic paralysis may be said to occur in about 15 per cent. of diphtheria cases. Accuracy in regard to this is not possible, as a number of such cases undoubtedly develop after the patients have passed from observation. The symptom usually occurs during the stage of convalescence. It may recur as early as the second day, and even after a month.

The cases are usually divided into a discrete or local form and severe or general form. The first, by far the more frequent, usually occurs earlier in the disease than the latter. The palatal muscles are those

most often involved, and even when the paralysis develops into a general one the palate is, as a rule, first affected. The first symptom noted is that the children appear to have difficulty in swallowing, and that liquid food causes an attack of spasmodic coughing and returns through the nose. On inspecting the throat it is seen that the uvula hangs down in a relaxed condition and does not respond to stimulus. This condition is recovered from in a few weeks. There is slight danger of an aspiration pneumonia being caused by it.

In the general form of paralysis the involvement of other groups of muscles usually follows that of the palate. The muscles of the pharynx and larynx usually come next in frequency, and then those of the lower extremities and the eye. The patellar reflexes are regularly lost, and there may be paresthesia or complete anesthesia of the limbs. The children, if allowed out of bed, either walk awkwardly with a shuffling gait or are entirely unable to stand. Any or all of the ocular muscles may be affected on one or both sides, in consequence of which there is ptosis, strabismus, hypermetropia, myopia, and inequality of the pupils. The muscles of the upper extremity are less often involved than the lower. When this takes place the paralysis is usually total, with absolute loss of muscular power; the patient not able to sit up or support the head, to speak, or to swallow.

Paralysis of the diaphragm usually occurs in connection with the involvement of other muscles. The respiration is purely thoracic and carried on by voluntary effort. During inspiration instead of the normal bulging of the abdomen there is a recession in this region, and with contraction of the chest the abdomen bulges. There is a real and painful dyspnea, and much anxiety on the part of the patient; the respirations are shallow and irregular. The prognosis in these cases is not good on account of the fact that cardiac paralysis is not an infrequent accompaniment. The latter may occur at any stage of the disease, but is apt to be delayed until convalescence is well established. It occurs as a part of a general paralysis or by itself.

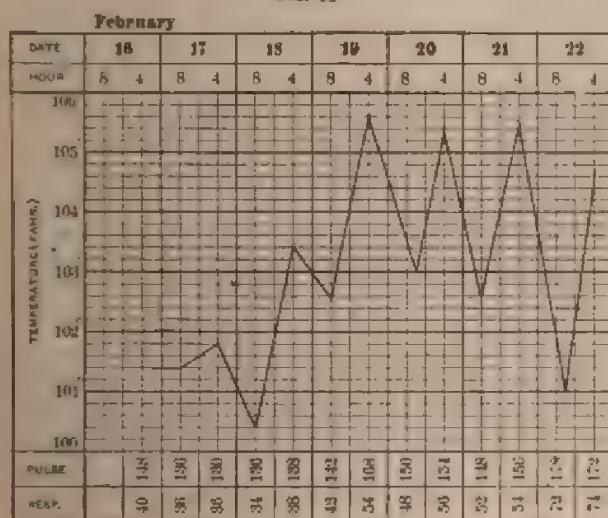
The involvement of the pneumogastric nerve is shown by vomiting and abdominal pains. In the milder form the pulse may be only weak or irregular, with a tendency to syncope. In the severe form the pulse may be very slow or very rapid, markedly irregular, thready, or intermittent. Various murmurs are heard over the precordium. The respiration is rapid; the patient is terribly anxious, tossing about, lying apparently lifeless. Sudden death may take place at any time, either with or without previous exertion. The prognosis in the severe forms is not good.

The paralyses, referable to the cerebrum, are the result of hemorrhage, embolism, or thrombosis, due, as already pointed out, to a change in the character of the blood. They usually occur during convalescence.

*Pulmonary Lesions.* It may be stated as a very general rule that there are few fatal cases of diphtheria which do not show the presence of pneumonic lesions as a contributing or direct cause of

This complication occurs very much more frequently in hospitals and institutions than outside of them, in children under one year of age than in older ones, in mixed infections than in pure diphtheria, in aratral cases, and especially those which have been operated on, even when the disease is located elsewhere; much more frequently in winter and spring than in the warm months, and in those institutions in which no attempt is made to isolate cases having this complication from those who have not, and finally more often in cases not treated by antitoxin or treated late in the disease than those who are so treated at an early stage. Thus it may be seen that any statistics as to the occurrence of complicating pneumonia are of little value unless the above factors be taken into account (Fig. 82). The symptoms do not differ from those of secondary bronchopneumonia complicating other

FIG. 82



Diphtheritic bronchopneumonia.

acute infectious diseases. There is an increase in the respirations, which are usually 50 or more to the minute, together with an increase in the pulse rate and a rapid rise in temperature. The respiration-pulse ratio approaches one to three or one to two. The prostration is increased.

In laryngeal cases the cyanosis is increased; the physical signs may be obscured by the transmission of sounds to the chest wall from the larynx, especially in intubated cases; so that the diagnosis must be based rather on the symptoms than the results of physical examination. A greater part of one or both lobes, generally the lower, is usually involved in severe cases. There is usually also more or less pleurisy, especially in the more chronic cases.

Bronchitis is a rather frequent accompaniment of diphtheria. Its importance is due to the fact that, particularly in younger children, it



shows a great tendency to spread downward to the smaller bronchi and air vesicles. Emphysema of a vesicular type is occasionally seen at autopsy, especially in operated laryngeal cases and when pneumonia is present in other parts of the lungs.

**Diagnosis (Clinical).**—There are a number of conditions so closely resembling true diphtheria that no matter how great the experience of the physician it will not enable him to arrive at a correct diagnosis save by a bacteriological examination in the more obscure cases. On the other hand, the great majority of cases of laryngeal and pharyngeal diphtheria may be diagnosed on the local and clinical symptoms, and it is to be remembered that, in a disease in which every hour's delay in administering specific treatment adds to the patient's danger, it is not always advisable to wait for the result of cultures.

The clinical diagnosis of pharyngeal and nasal diphtheria is based on the character of the membrane already described, on its tendency to spread to adjacent parts, the condition of the pulse and temperature, and evidences of toxemia. Follicular tonsillitis, especially when the individual areas tend to coalesce and resemble a membrane, may not infrequently be confused with diphtheria. In the former, however, both tonsils are usually involved simultaneously, there is no tendency to spread to other parts, the individual crypts of the tonsil, filled with cells and detritus, may usually be made out, and when, by the coalescence of such detritus, there is an apparent membrane, the latter may readily be brushed off, as it is not adherent. The constitutional symptoms come on very much more quickly and violently than is the rule in diphtheria; there is sudden high fever, headache, pain in the joints, and the patient feels very ill. It rarely occurs in very young children. There is a rare form of true diphtheria which so closely resembles follicular tonsillitis that a clinical diagnosis is not possible. Non-diphtheritic membranous tonsillitis occurs in the majority of cases in the course of the acute exanthemata, notably scarlet fever and measles. It is due to the pyogenic cocci and may occur as a primary disease. When it occurs secondarily to the exanthemata, its true nature may be suspected, but as true diphtheria not infrequently complicates these diseases too much reliance should not be placed on the clinical diagnosis, and cultures should be invariably taken. In the primary cases it is not possible to distinguish the condition from that of true diphtheria. The membrane is usually seen on the tonsil and shows perhaps less tendency to spread than in the latter disease. The symptoms do not serve to make a differential diagnosis.

Circumtonsilar abscess occasionally bears some resemblance to diphtheria, especially as there may be more or less membrane on the tonsil due to the growth of pyogenic cocci. The clinical picture, however, is generally fairly typical. The tonsil usually at one side is pushed toward the middle line; it is congested as well as the mucous membrane above it. There is difficulty in opening the mouth and the speech resembles that of a person speaking with his mouth full. Evacuation of the pus is followed by immediate relief.



Herpes, sprue, and ulcerative stomatitis may occasionally be confounded with buccal diphtheria. In cases of doubt the diagnosis should rest upon culture. The rather rare ulceration of the tonsil due to the bacillus of Vincent need only be referred to as occasionally mistaken for diphtheria. This together with tuberculous and syphilitic lesions are to be differentiated by the history of the case and finally by culture.

*Diphtheritic Croup.*—No description, written or oral, however graphic, can take the place of actual observation of one or more cases of this condition in enabling the physician to recognize it almost at a glance. So characteristic are the symptoms, that only lack of familiarity with them can excuse a failure to recognize them after the signs of disease are well marked. The symptoms have already been described. Those of most importance in arriving at a diagnosis are the character of the cough; the somewhat slow, insidious, and, notwithstanding frequent remission, steady increase in the symptoms of stenosis; the presence, as a rule, of membrane on the tonsils or pharynx, and, when stenosis is well established, the recession at the epigastrium and clavicles.

Cases of membranous croup due to other organisms than the Klebs-Loeffler bacillus are occasionally reported; usually they occur as a complication of scarlet fever or measles or other exanthematous diseases. Doubtless this condition has been more frequently diagnosticated than the known facts would seem to justify. The uncertainty of early laryngeal culture has already been pointed out, and in my opinion this fact is accountable for many of the cases of membranous laryngitis being reported as non-diphtheritic. In membranous laryngitis without membrane in the pharynx subsequent cultures will almost invariably show the Klebs-Loeffler bacillus, even if the first are negative or show the presence of a few cocci. While this condition undoubtedly is occasionally met with, nevertheless the diagnosis of membranous croup due to another organism than the diphtheria bacillus should be made with the greatest reservation.

Catarrhal croup may be mistaken for true diphtheritic laryngitis. In the former, however, the attack comes on very suddenly, either without a history of previous illness or one of mild catarrhal trouble or indigestion shortly before. The child is awakened, usually at night, with sudden symptoms of suffocation, a characteristic barking cough, partial aphonia, and intense anxiety. If the true condition be suspected, the administration of an emetic, a hot mustard bath, with steam inhalation, will promptly relieve the symptoms. The next morning the child will be in comparatively normal health, though the attack is apt to recur the following night, and sometimes lasts for a night or two more.

Retropharyngeal abscess is occasionally mistaken for laryngeal croup. In the former the child's head is thrown back, the mouth held open, the voice likened to the quacking of a duck. On digital examination of the throat there will be found at the back of the pharynx, on the middle line or at one side, a characteristic fluctuating tumor. Evacuation of the pus produces immediate relief.

Bronchopneumonia is not infrequently mistaken for membranous laryngitis, and intubationists are not uncommonly called upon to operate on such cases. There is dyspnea in both, cyanosis, recession of the soft parts of the chest, and evidences of toxemia. Here, however, the resemblance, as a rule, ends, and a knowledge of the symptoms and signs of the two diseases should enable one to avoid mistakes in diagnosis.

*Nasal Diphtheria.*—This disease may be suspected when a membrane cannot be seen in the nares, if a nasal discharge persists with marked obstruction, and especially when the former is freely mixed with blood and pus and causes excoriations of the nostrils and lips. Nevertheless, the diagnosis can only be confirmed by culture taking. In a postnasal case with enlarged lymph nodes if the child will allow a laryngoscopic examination it will help clear up the diagnosis. Severe acute cases usually follow a pharyngeal or tonsillar diphtheria, and are, therefore, not difficult to recognize.

**Diagnosis (Bacteriological).**—From what has been said of the protean character of the symptoms of diphtheria, it should be evident that for purposes of exact diagnosis the clinical symptoms must in many cases be secondary in importance to the knowledge obtained by bacteriology. When it is essential to make an immediate diagnosis the direct method may be employed, the results of which, however, are by no means always satisfactory. For this purpose a forceps or regular culture swab, wound with absorbent cotton, is passed over the suspected surface, removing, if possible, a bit of membrane. A drop of clean water is then placed on a cover-glass or microscopic slide, and a smear made on the surface. This is passed through the flame in order to fix the specimen and stained with Loeffler's alkaline methyl-blue solution, dried, mounted, and examined. The diphtheria bacilli, if present, do not resemble closely those seen in cultures. Here or there one or two may be found in the fibrin and detritus; they are short rods, often swollen slightly at one or both ends. Cocci of various kinds are usually present. If the bacilli are not found after a careful search, but a good many cocci are seen, it is fairly good evidence that the disease is not true diphtheria. If, on the other hand, bacilli are present, it cannot be said from their morphological character that they are positively of the Loeffler variety. The appearance is not typical and the staining qualities are subject to much variation. It is a safe rule with the presence of membrane to regard bacilli in a direct culture, even if atypical, as those of true diphtheria, if there be occasion for immediate treatment.

In taking cultures a sterile cotton swab is applied thoroughly to the affected surface, care being taken to ascertain that no antiseptic has been used for a number of hours previously. In suspected laryngeal diphtheria, without apparent pharyngeal involvement, it is advisable to apply the swab if possible directly to the interior of the larynx. The mucus and portions of membrane thus obtained are rubbed gently and thoroughly over the surface of a tube of Loeffler's blood serum, which

itself has been rendered sterile. The cotton swab is then removed, passed through the flame, or returned to its individual tube and plugged with absorbent cotton. The culture tube, after being similarly plugged, is placed for twelve hours in an incubator with the temperature kept at about 37° C. (99° F.).

For the examination of cultures, a sterile platinum loop is passed over the surface of the culture medium so as to remove a number of colonies. A drop of sterile water is placed on slide or cover-glass, the bacterial contents of the loop washed off in it and smeared over the surface. It is then dried in the air, fixed by passing it through the flame, and stained from five to ten minutes with the Loeffler solution, washed off in water, dried, mounted, and examined, preferably with an oil-immersion lens. The Neisser stain may be used if desired, but it is doubtful if anything more may be learned from it than the simpler methyl blue.

Cultures which show pure cocci may be regarded as conclusive evidence that the case is not one of diphtheria. When the bacilli are few in number and are atypical, unless the clinical evidence points strongly to the existence of diphtheria, secondary cultures should be taken. Furthermore, when there is strong clinical evidence of diphtheria, especially in laryngeal cases, negative cultures should be disregarded in the presence of urgent symptoms and specific treatment begun at once.

**Prognosis.**—No disease, unless it be bronchopneumonia, is so uncertain in its outcome as diphtheria, and in each case a number of factors have to be considered in forming a prognosis. Even then the practitioner will often see his most hopeful cases terminate fatally, and the seemingly most hopeless go on to ultimate recovery. No more important element enters into the prognosis than that of the patient's age. Children under one year possess a certain immunity to this as well as most other infectious diseases, and especially nurslings under six months. When such a child does contract the disease the prognosis is not favorable.

A general idea of the mortality according to age is afforded by the statistics of the Boston City Hospital. All the cases were treated by antitoxin. In children under five years death occurred in about 20 per cent. of the cases, in those from five to ten in about 8 per cent., and from ten to fifteen 3 per cent.

The individual constitution of the patient is an important factor in influencing the outcome of the disease. Those who are anemic, rachitic, the subjects of lymphatic hypertrophy and digestive disturbances show less resistance to the disease than those previously in good health.

Institutional and tenement-house cases show less favorable results than those taken from more healthful surroundings. Pure pharyngeal cases may be considered the most favorable, with the exception, perhaps, of the more chronic nasal ones.

Laryngeal cases, especially when operated on, justify the least favorable prognosis. In hospital practice the death rate of the latter has,



by the use of antitoxin, been reduced from over two-thirds to one-third or less. In private practice the death rate is much lower. The mortality of tracheotomized cases is somewhat higher than those intubated.

Mixed infections, from their greater liability to be followed by complications, justify a less favorable prognosis than cases of pure diphtheria. The death rate in cases complicating the exanthemata is for this reason greater than in primary cases. The time of beginning antitoxin treatment is of all importance in forming a prognosis. Thus the death rate varies roughly from 5 per cent. in those treated in the first twenty-four hours to 35 or more in those in which it has been delayed for four or five days. In other words, after the fourth day, the death rate approaches that of pre-antitoxin days. The cases occurring in the winter are more likely to be followed by pulmonary complications than those of the summer months. In general, marked toxemia, as evidenced by restlessness, stupor, delirium, rapid and irregular pulse, paralysis, and kidney involvement; the occurrence of pneumonic lesions, marked involvement of the lymph nodes and rapid spread of the membrane, all justify an unfavorable prognosis.

Diphtheria affecting other mucous membranes than those of the nose, pharynx, and larynx, as well as that occurring on abraded cutaneous surfaces, rarely terminates fatally. In that affecting the conjunctiva, when properly treated at an early stage, the prognosis in regard to local after-effect and loss of sight is equally good.

**Prophylaxis.**—Children with tendency to hypertrophy of lymphatic tissue of the nose and pharynx should be especially careful to avoid exposure, and these conditions should receive proper attention. All cases of diphtheria should be isolated as perfectly as possible; no other than the nurse or member of the family acting in that capacity and the physician should be allowed in the room, the doors of which should be kept closed as much as possible. A large vessel (pail to be preferred) partly filled with a carbolic solution of a strength of 1 to 20 should be kept in the sick-room. Articles of wear, handkerchiefs, towels, cotton swabs, etc., upon which discharges from the nose or mouth have been caught, should be soaked in this solution for several hours, after which they may be removed, boiled, and washed in a regular way. Bed-clothes and surfaces soiled by discharges should be disinfected immediately. Utensils, dishes, cups, etc., should be kept for the exclusive use of the patient and not sent from the sick-room. The room should be thoroughly aired, and if more than once can be utilized the patient should be removed once a day while the adjoining room is swept and cleaned, the floor being previously covered with wet paper or tea-leaves, and the sweepings afterward burned and disinfected. All unnecessary furniture, especially of the upholstered variety, should be removed at the beginning of the illness.

The patient should not be allowed out of quarantine until culture taken from the site of the lesion no longer show the presence of *Klebsiella-Loeffler* bacilli, even though this does not occur for several weeks. When



the patient is ready to be discharged, he or she should be given a thorough bath, the hair, face, and body thoroughly washed with soap, and afterward dressed in clean clothes which have not been exposed to contamination. When obliged to leave the sick-room, the nurse should change the outer clothes or remove her contagion robe, wash the hands, first in soap and water, afterward in bichloride of mercury solution of the strength of 1:1000. The face should also be washed in a weaker solution. Nurses who consider themselves susceptible should receive immunizing doses of antitoxin during their attendance on diphtheria cases. A gargle of boric acid, listerine, or Dobell's solution may be used with advantage several times a day. The physician should wear a contagion robe before entering the sick-room and leave it in the room on his departure. It should completely envelop the clothes, the neck, and wrists. In addition it is well to wear a cap covering the hair (see Fig. 90). Before leaving the house the hands, face, and beard should be thoroughly washed in a disinfectant.

When the patient is ready to be dismissed from quarantine the room should be disinfected as follows: The walls should be rubbed down with bread, damp cheese-cloth, or, when possible, washed in bichloride of mercury solution 1:1000. The woodwork, furniture, and floor should be washed with the same solution. Books, toys, etc., should be burned. Steam disinfection may be used for upholstery of all kinds. A general disinfection may be performed by the use of formaldehyde vapor, preferably under pressure, or sulphur, the doors and windows being previously plugged with absorbent cotton.

For the prevention of diphtheria in those who have been exposed to it no means are so entirely satisfactory as immunization by antitoxin. The immunity conferred, though but temporary, three weeks or less, nevertheless gives sufficient time for the original course of infection to disappear and thus prevent the local spread of the disease. This is especially important in institutions during an outbreak of diphtheria, and also during epidemics of other infectious diseases, notably measles, upon which the former is so apt to be engrafted. During measles epidemic in children's institutions it is now the regular custom to give immunizing doses of 500 units to all the children affected. The result has been a marked decrease in the death rate from diphtheria complicating measles. The following taken from a recent pamphlet issued by the Department of Health of New York City speaks for itself, and should be conclusive proof of the value of immunity conferred by antitoxin:

From January 1, 1895, to January 1, 1903, immunizing injections of antitoxin were administered to over 13,000 individuals by the inspectors of the Department of Health, and by physicians (free cases only). Of these individuals 40 (0.3 per cent.) contracted diphtheria of a mild type, one case only terminated fatally.

The records of the Division of Bacteriology show that during one year alone 682 cases of diphtheria occurred in New York City, which were secondary to an original case in the same family. Under "Sec-

ondary" are included only those cases which occurred at least twenty-four hours after and within thirty days of the primary case. Of these 682 cases 61 died, a mortality of 8.9 per cent. *Had these 682 individuals received antitoxin when the physician first visited the family, probably not one of them would have contracted the disease.* The above figures represent only a fraction of such secondary cases occurring in New York City.

In private cases it should be routine practice to give immunizing doses of antitoxin at least to all the young members of the family who have been exposed. The only ill effects are an occasional rash, which causes some discomfort and a slight rise in temperature. The security obtained more than repays for the inexcusable dread which some persons exhibit of inoculating a healthy person for any purpose.

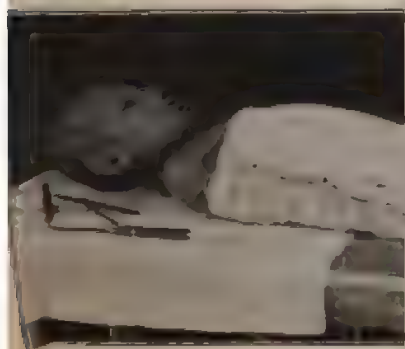
**Treatment.**—There is but one method of successfully combating diphtheria after it has once occurred, now recognized by practically all physicians throughout the civilized world, namely, by the use of properly prepared antitoxin given in sufficient dosage and as early as possible in the course of the disease. Since its use has become general, one after another of the remedies formerly regarded as possessing a specific action have passed into disuse. Certain adjuvant measures are still of great importance in order to promote the comfort of the patient, preserve the strength and diminish the risk of complications, and to increase the chances of recovery when these have occurred. The patient should be put in bed and kept there during the entire course of the disease. The room, or preferably two adjoining rooms, should be sunny and always well aired. Running water should be included if possible. Patients having complications, especially pneumonia and septic conditions, should on no account be treated in the same room as one whose case is not so complicated. This rule should be, but unfortunately is not, applied to hospitals as well as to private patients. Easily digested food should be given at two or three hours' intervals and in small quantities, though there is but little danger of a patient overeating. Milk and the various beef preparations are often more readily managed than solid food. Semisolids are usually well taken. Gavage may be used in young children in intubated cases, and in those in which the condition of the throat prevents the taking of food. For this purpose the child should be wrapped as described for intubation (Fig. 83) and the tube, a catheter attached to a glass funnel, passed through the nose or mouth into the esophagus. The necessary quantity of milk is then poured in, and the tube before withdrawal pinched between the thumb and forefinger to prevent the last few drops from entering the larynx and exciting an attack of coughing, a desirable precaution in intubated cases.

Rectal feeding may be used as a temporary expedient when food cannot be readily swallowed or is not retained. The bowels should be kept open, preferably by enemata. If reduction of the fever is necessary it should be accomplished by sponges or packs at 85° to 95° F.

All severe cases with evidences of marked toxemia, and especially those with sepsis, require constant stimulation, as do those in which

the heart action is weak and irregular. Whiskey and brandy of the best quality, in doses of 0.60 c.c. (10 drops) or more every two hours in 15 c.c. (half an ounce) of water, may be given to a child of a year for an indefinite time. For older children 60 c.c. (2 or more ounces) may be given in the twenty-four hours, well diluted. Strychnine in doses of 0.00065 gm. (gr.  $\frac{1}{1000}$ ) every two hours may be given to the youngest child, and twice as much to one three years or over. The symptoms of poisoning should be carefully watched for. Strychnine may be given hypodermically in the same doses, or nitroglycerin in doses of 0.00016 to 0.00065 gm. (gr.  $\frac{1}{1000}$  to  $\frac{1}{1000}$ ), depending on the age of the child, when rapid stimulation is called for. Other cardiac stimulants, as digitalis, may be indicated in certain conditions of the heart. Their effect is more lasting, but they have a tendency to upset the stomach. Sedatives, such as bromide of sodium, are often required. They are to be used when the child is restless and wearing out its strength by tossing about, and when there is evidence of cardiac involvement, in which

FIG. 83



Method of wrapping the patient for operative or local treatment and gavage.

perfect quiet means so much for the ultimate recovery of the patient. Sedatives may also be used before removing the tube in intubated cases, and the cannula in tracheotomized cases, in order to diminish the probability of having to reinsert it; in laryngeal cases, which have had antitoxin for the purpose of overcoming a spasmodic attack of dyspnea, sedatives may be given, intubation put off as long as possible, and the antitoxin given an opportunity to accomplish its purpose.

No drug is so certain in these various conditions as morphine given hypodermically in doses of 0.00324 to 0.0054 gm. (gr.  $\frac{1}{200}$  to  $\frac{1}{100}$ ), and repeated if necessary at two-hour intervals. As a general sedative when a rapid effect is not necessary, Dover's powder in doses of 0.03 to 0.06 gm. (gr.  $\frac{1}{2}$  to 1) or more repeated is of good service.

Emetics are of value in certain conditions, although their use is much more limited to-day than formerly. They may be used to clear the throat and larynx of thick mucus and membrane, or when it is not possible to intubate at once in laryngeal cases requiring the operation.



There is rarely occasion to make use of them except at the beginning of the disease. For this purpose syrup of ipecac in full dosage is the least harmful and is generally effective. As a means of making a differential diagnosis between a case of catarrhal croup and true diphtheria an emetic, together with the other measures for the treatment of the condition already described, is perfectly justifiable. The stenosis of true croup quickly returns, while the catarrhal condition is generally relieved, at least for many hours.

**Local Treatment.** At the present time local treatment with the object of directly affecting the diphtheritic process has been almost universally abandoned, and forcible removal of membrane and the applications of strong bactericidal remedies have been proved to be not only useless, but in many cases actually harmful. A cleansing irrigation with mild, bland solution for the purpose of removal of already detached membrane, together with thick mucus and pus, and reducing the local congestion are of the greatest value. For this purpose there is needed an ordinary fountain syringe holding two quarts, or irrigator of glass or agate ware, an olive-tipped, hard-rubber or glass nozzle for use in the nose, and a long, hard-rubber tip for use in the throat. A solution of common salt of strength of 4 gm. to 0.6 litres (a teaspoonful to a pint); a saturated solution of boric acid, or one of the ordinary mouth washes, such as listerine well diluted, is preferable to most other and stronger solutions. A temperature of somewhat over 100° F. for ordinary cases, or one 125° F. or more with the object of reducing congestion is to be used. The receptacle should be placed four or five feet above the patient's head, the latter wrapped in a sheet as shown in the illustration (Fig. 8) over which is pinned a rubber blanket closely round the neck. The patient is put on the side on a table, its head lying on a Kelly pad, which should drain into a solution of carbolic acid. The head is held firm against the table with the left hand. The nozzle of the syringe is then placed in the upper nostril and a small amount of water allowed to flow, after which it is momentarily removed and the patient permitted to take a breath or two to be reassured. The irrigation is then continued until the result of the washing is a perfectly clear fluid. By this means large nasal plugs of fibrin are often removed, which otherwise would serve to obstruct respiration and by their decomposition serve as a source of infection by various organisms. For the irrigation of the mouth, which is usually performed, when necessary, directly after that of the nose, the special nozzle is passed gently between the cheek and teeth, the water allowed to flow, and, as the mouth opens, the tip of the nozzle is gradually passed to the middle line, the tonsils and pharynx being in this way cleansed from thick mucus, pus, and pieces of detached membrane.

The operation may be repeated every four hours in ordinary cases and every two hours in those in which there is a great deal of nasal discharge, especially of a purulent variety.

While there is a difference of opinion in regard to the propriety of this procedure in certain cases, I believe that there are practically



contraindications, and that there is no good evidence that disease of the middle ear is more apt to follow cases which are irrigated than those which are not. Furthermore, there is no good reason why intubated cases with nasal involvement should not be so treated, and while the operation occasionally causes an attack of coughing, which may result in the expulsion of the tube, the latter may be immediately reinserted. The value of irrigation in such cases far outweighs the inconvenience

FIG. 84



Irrigation of the nose.

of this occasional occurrence. Finally, there is no evidence that aspiration pneumonia is ever caused by the procedure.

A less efficacious method of cleansing the nose and throat is that by means of an ordinary syringe or bulb syringe. Too much force is apt to be used, it is less agreeable to the patient, and the result is far less satisfactory. In cases of cardiac paralysis great care should be taken to avoid exciting the patient, but as this condition usually occurs late in the disease the indication for irrigation is not often present.

Local applications for the relief of spasm and pain and reduction of swelling are occasionally of service. Steam inhalations by means of a croup kettle, the child being placed under a canopy, may be continued for from one-half to one hour at a time. Hot, thin, flaxseed poultices placed on the throat, and immediately removed on cooling, seem to afford relief in these conditions. The steam, however, should on no account be kept up for any great length of time to the exclusion of fresh air, and the poultices not used oftener than at intervals of two or three hours.

In conjunctival diphtheria, in addition to the use of full doses of antitoxin, the treatment should in general be that of a purulent conjunctivitis, namely, ice-cloths applied every few minutes to reduce the swelling and congestion, mydriatics, atropine (or cocaine), constant separation of the lids and washing out of the eyes by means of a medicine dropper, boric acid or other mild solution, and the application of a drop or two of nitrate of silver solution several times a day.

At the Boston City Hospital the use of the red or yellow iodide of mercury as a local application to the lids, 0.06 gm. to 30.00 gm. (1 grain to 1 ounce vaselin), is believed to have been of benefit.

**Antitoxin.** In 1893 Behring may be said to have established the real value of antitoxin in the treatment of diphtheria. Previous to this he, with other experimenters had made tentative trials of it, but in an imperfect manner. It is not remarkable that since the introduction of this specific remedy there has risen opposition to its use from time to time. This has rested upon reports of sudden death after its administration, septicemia, tetanus, local infection, and of negative results following its use. It is undoubtedly true that in the early days of antitoxin the serum was not always what it should have been. It was not sufficiently concentrated, nor always pure or properly preserved. The proper dosage was not definitely known, nor were the limitations to its efficacy appreciated. It is not, of course, possible to investigate the truth of many of these reported mishaps, but it may be set down as an indisputable fact, deduced from hundreds of thousands of cases in which antitoxin has been administered and its effect carefully watched, that in no case has death been caused by a properly prepared pure, fresh, serum.

Death can occur and has occurred, and alarming symptoms have followed an infected serum, one that has not been properly preserved and one injected without proper antiseptic precautions. Sudden death occurs in diphtheria with or without the use of antitoxin; that it should be attributed to the remedy and not the disease in a certain number of cases is not difficult to understand. There is, however, no such good reason for the belief that antitoxin *per se* is dangerous to life. Certain symptoms, however, that are not dangerous to life, very often follow its administration, and they are now generally recognized and will be noted later. The production of antitoxin should be under strict municipal control even if the actual manufacture be left to private concerns, as the greatest possible care is essential.

*Effect of Serum Treatment.*—The following table just published by the Department of Health of New York City may be taken as fair:

representative of the effect of antitoxin upon the general mortality in a large number of cases. These, of course, include hospital cases as well as those treated in private practice, cases dying of complications and those in which the antitoxin was not administered until very late in the disease, and, in not a small number, where antitoxin was omitted for various reasons, such as the wish of the parents or disbelief in its value by the attending physician.

TABLE SHOWING NUMBER OF CASES, DEATHS, AND MORTALITY PER CENT. OF DIPHTHERIA IN THE BOROUGH OF MANHATTAN AND THE BRONX, FROM 1893 TO 1904, INCLUSIVE:

Period.	Cases.	Deaths.	Mortality per cent.
1893 . . . . .	7,021	2588	36.4
1894 . . . . .	9,641	2870	29.7
1895 . . . . .	10,353	1376	19.1
1896 . . . . .	11,399	1763	15.4
1897 . . . . .	10,806	1590	14.6
1898 . . . . .	7,563	923	12.2
1899 . . . . .	8,240	1067	13.1
1900 . . . . .	8,364	1121	13.4
1901 . . . . .	7,726	1257	15.9
1902 . . . . .	10,429	1112	10.9
1903 . . . . .	11,662	1302	11.2
1904 . . . . .	12,383	1272	9.57

At the New York Foundling Hospital the mortality rate from all cases of diphtheria complicated and uncomplicated, primary and secondary, to other infectious diseases, operative and non-operative, is 9 per cent. in 300 cases.

It is possible for many physicians to make a far better showing than the above in cases taken from their private practice.

Probably a death rate of 4 or 5 per cent. will fairly represent the results of uncomplicated cases treated on the first day. While the general mortality has decreased so markedly the death rate among infants is still very high, and has not apparently been reduced in the same proportions as that among older children, a fact which is due not only to the great susceptibility of infants to the specific toxin, but to the frequency of pulmonary complications.

*Effect upon Laryngeal Diphtheria.*—In these cases the benefits of antitoxin may be seen in two ways: first, by reducing greatly the number of cases which require operative interference, and second, by reducing the death rate in operative cases.

The great extent of membrane formerly seen in the larynx and trachea is now seldom met with.

Furthermore, the time during which a tube must be worn continuously has been notably decreased, and multiple reintubations are less frequently required. Before the days of antitoxin nearly every case of laryngeal diphtheria progressed to the stage where operative interference was necessary; to-day only one-half of such cases require it. Without antitoxin death occurred in about two-thirds of the cases operated on. With antitoxin the death rate is less than one-third, and it is possible to quote a number of physicians who have had a dozen



or more cases in private practice without a death. The statistics in regard to tracheotomy are only less favorable than those of the bloodlet operation.

*Effect on the Occurrence of Complications.*—In regard to the nervous system it has been shown experimentally and corroborated clinically that antitoxin administered at the time, or shortly after the diphtheritic toxin had become active, provided it is given in doses sufficient to neutralize the latter, regularly prevents the occurrence of paralysis. So sensitive is the nervous system to this particular poison that even delay of twenty-four hours greatly adds to the probability of occurrence of nervous symptoms, and after the second day the effect of antitoxin in this regard is practically negative.

The same rule applies to the cardiac symptoms of diphtheria. Nephritis is not a common complication of pure diphtheria. It has been shown that it is less likely to occur with the use of antitoxin than without it. Upon the occurrence of complications, due to associated organisms such as the streptococcus, pneumococcus, and staphylococcus, antitoxin has a real but indirect effect in that it shortens the course of the diphtheria and restores to the normal, at an earlier date, the affected mucous membranes; so that there is less opportunity for the production of complicating lesions by these organisms, and, with the early administration of antitoxin, bronchopneumonia, local suppurative conditions and general sepsis are less frequently observed.

When diphtheria is implanted upon another infection in which pyogenic germs play an important part, as in scarlet fever and measles, the benefit of diphtheria antitoxin is greatly reduced, and it is in this class of cases that preventive measures are of so much importance. Children suffering from one of these diseases and exposed to diphtheria should never fail to receive full immunizing doses of antitoxin at once.

*Administration.*—There are practically no contraindications to the use of antitoxin when the diagnosis of diphtheria is once established. In those cases which have existed from five to seven days or more when first seen by the physician, it may be questioned whether the use of antitoxin is called for in view of the fact that its power will be so greatly diminished by the delay in administering it. In severe late cases, should always be given even if it be regarded as a last resort.

In all cases of diphtheria in children under two years of age, however mild or at whatever site the lesion; in all croup cases suspected to be due to diphtheria, and in all doubtful croup cases; in every case in which there is a membrane, with evidence of toxemia, unless positive known to be of non-diphtheritic origin, in all suspicious eye cases, and antitoxin in full doses should be given at once without waiting for the result of culture, and without placing too much reliance upon the direct bacteriological finding by the method previously described.

In mild pharyngeal cases in older children; in membranous pharyngitis or tonsillitis occurring with scarlet fever or measles; in chronic suspicious nasal cases; in anogenital cases; in typical follicular tonsillitis; in catarrhal angina which, on account of the exposure of the patient



to diphtheria, may be regarded as suspicious, it is allowable to wait for the result of cultures, in the absence of alarming symptoms, but in every case of doubt when such symptoms are present, antitoxin should be given at once. Thus, while the vast majority of cases of membranous angina which occur in scarlet fever and measles are due to pyogenic cocci and those of tonsillitis with a typical follicular distribution due to the same organism, yet the Klebs-Loeffler bacillus is not infrequently found associated with them under these conditions.

*Dosage.*—In ordinary mild cases of pharyngeal or nasal diphtheria 2000 to 4000 units should be given. In severe cases, at the same location, twice as much. The smaller the child the smaller is the dose required, but it is better to give too much than too little in any case. In laryngeal cases 5000 to 10,000 units, depending not so much on the symptoms as on the age of the patient, should be given. This dosage should be repeated in twelve hours if the local conditions appear less favorable, and in laryngeal cases if the symptoms of stenosis fail to show an amelioration. A third dose is sometimes required. It is very doubtful if the huge dosage of 4000 to 50,000 units or more has produced any better results than in those of the size just enumerated. For purposes of immunity the dose should be from 500 to 1000 units, depending on the age of the child and on the probability of its contracting the disease. In eye cases 5000 units or more should be given. Any syringe holding 10 c.c. which can be boiled may be used. A small needle is preferable. The skin at the site of injection should be cleaned with soap and water, alcohol, and bichloride of mercury solution. The tissues below the scapula on the outside of the thigh or buttocks may be selected. The serum should be injected deeply, and, after withdrawing the needle, a piece of sterile gauze or cotton placed over the wound and held in place by a piece of adhesive plaster or collodion.

*Clinical Effects of Antitoxin.*—A few hours after injection, seen most typically in cases of pure diphtheria of the tonsil, the membrane begins to swell, and later its edges become loosened from the underlying mucous membrane and curl up. Very soon it detaches itself *en masse*, or, more often, in small pieces. The detachment takes place in from twenty-four hours to four days. In nasal cases membranous casts of the nares are loosened and come away in the irrigation fluid. In eye cases, especially those in which there is not mixed infection, the process is seen in its typical form and similar to that of the tonsils. In laryngeal cases, if the case progresses favorably, after a few hours the stenotic symptoms gradually disappear. If, on the other hand, the case goes on to intubation, and there is an appreciable amount of membrane present, the latter is either coughed up through the tube or may obstruct its lumen and causes autoextubation or removal of the tube. The moderate involvement of the lymph nodes of the neck seen in pure diphtheria rapidly subsides, the temperature and pulse decrease, and the toxemia soon disappears.

In mixed cases all these phenomena follow the use of antitoxin, but are less well marked. Thus the temperature and pulse rate may remain

high and the general condition of the patient not show the same improvement as in cases of pure diphtheria.

*Effect on the Blood.*—As shown by Ewing and others, antitoxin has the effect of decreasing the hyperleukocytosis caused by diphtheria. When this has not occurred the disease has often terminated fatally. Other observers have noticed a diminution of hemoglobin and red blood cells following the injection.

*Effect of Horse Serum.*—Certain clinical manifestations very regularly follow the injection of antitoxin. These are now recognized as being due wholly to horse serum and not to the antitoxin itself; for they have been shown to follow the injection of the former when in its natural state. The frequency of their occurrence as well as the severity of the symptoms depends to a great extent on the amount of serum injected, but it is also found that that of certain horses much more regularly produce them than others, and they are for that reason usually discarded. The greater concentration of the antitoxin has greatly diminished the number of cases in which these manifestations occur.<sup>1</sup> They are never dangerous to life, but are frequently the source of grave discomfort to the patient for the time being, and, therefore, to be avoided if possible. Various skin eruptions follow the injection of antitoxin in from 10 to 25 per cent of the cases. They occur from the second day to the third week or even later. The eruption may be confined to the site of inoculation or it may cover more or less of the face and body. In any event, the point of inoculation is usually the starting point. The most frequently observed eruption is a general erythema; the next most common an urticaria frequently seen in connection with the former. Others less common are scarlatiniform and morbilliform. Mixed varieties are frequently observed. The scarlatiniform and morbilliform rashes regularly occur at a later stage than the others. The duration of these various eruptions varies from a few hours to a day or two. They not infrequently disappear and return again. There is regularly a rise in temperature and pulse, an intense itching, and irritability of the skin; in some cases the eyes and face are intensely swollen; less often intense pain in the joints is observed, occasionally with swelling and redness. The fever reaches its height with the full development of the eruption and then rapidly subsides. The diagnosis of the scarlatiniform variety is often difficult as it very closely resembles true scarlet fever. The points on which it is based are the starting point of the rash from the point of inoculation, its very rapid and more general distribution, its evanescent character, and, very often, the lack of uniformity of the eruption; other varieties such as urticaria, occurring in other parts of the body. In cases of doubt the patient should be isolated.

<sup>1</sup> The Health Department of New York is now supplying a purified and concentrated diphtheria antitoxin. This product is extremely reliable and is not apt to produce rashes and other deleterious effects as the ordinary serum.

## INTUBATION.

Intubation for the relief of acute laryngeal stenosis was perfected after three years of experimentation by Dr. Joseph O'Dwyer, of New York in 1883. This operation has almost entirely superseded that of tracheotomy in America and on the continent of Europe. In England the older operation is still frequently performed.

**Intubation Instruments.**—The present tubes are made of vulcanized rubber on a metal frame, in six or more sizes corresponding to the age of the child. When in position they reach nearly to the bifurcation of the trachea. The retaining swell is of such a calibre that the cricoid constriction of the larynx keeps the tube in place under ordinary circumstances and yet allows of its ready expulsion when the lumen is blocked

FIG. 85



O'Dwyer's intubation tubes.

FIG. 86



Mouth-gag.

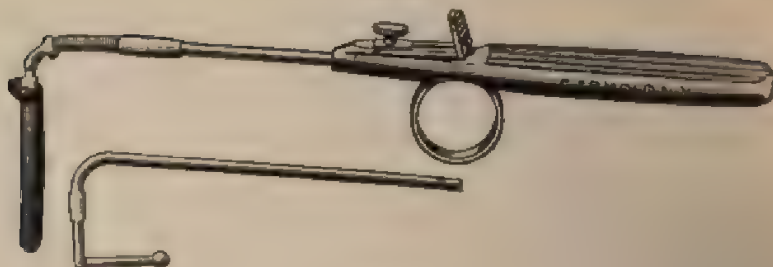
by loose membrane. The neck is made as narrow as possible and is gripped by the vocal cords. In order to avoid ulceration of the mucous membrane by pressure the head of the tube has been given a backward sweep and is somewhat thick, so that no sharp angle is presented to the base of the epiglottis. The end of the tube is blunt and well rounded off to prevent ulceration by the movement of the trachea over this part. Furthermore, it is advisable to use the smallest possible tube for the age of the child to diminish pressure at the cricoid constriction.

The general character of the tubes and instruments is shown in the illustrations (Figs. 85 to 89).

The tubes for false membrane are hollow cylinders in graded sizes of just sufficient length to reach beyond the cricoid constriction and are for temporary use only to allow the expulsion of the detached membrane when this is suspected to be present. They should on no account

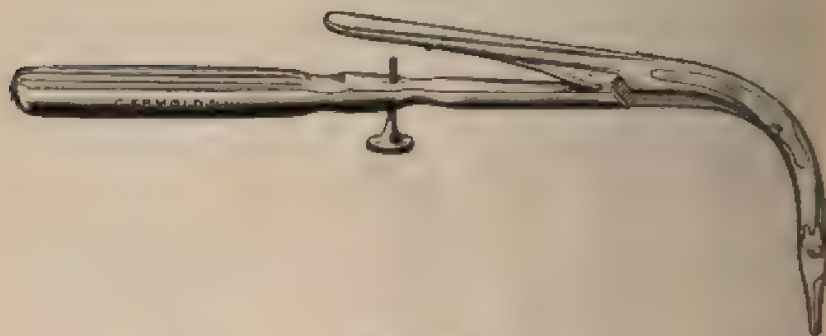


FIG. 87



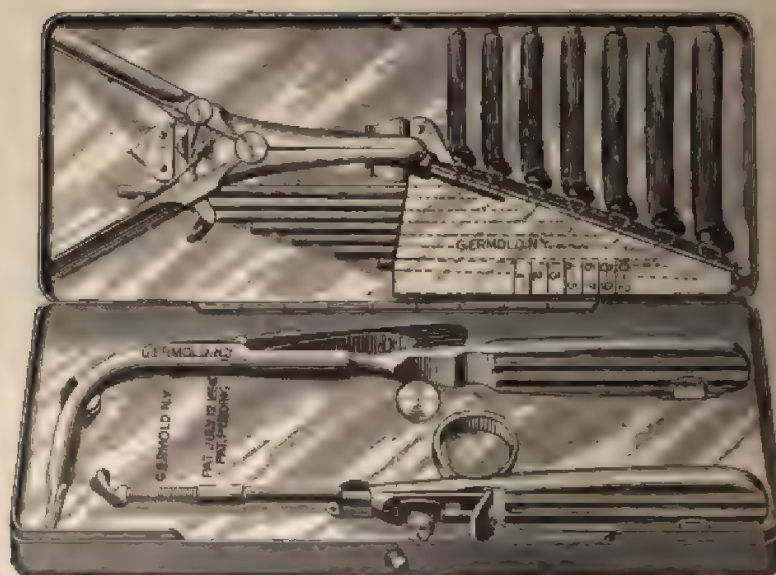
Introducer.

FIG. 88



Extubator.

FIG. 89



O'Dwyer's intubation set in case.



be left in position for longer than an hour or two, as, unlike the regular tubes, they are not adapted to the anatomy of the larynx.

Special tubes with built-up heads are occasionally used with the object of riding over the granulations caused by the disease and thus promoting their absorption.

FIG. 90



Intubation in the upright position. The left forefinger is on the epiglottis. The handle of the intubator is exactly parallel to the body. The tube is at the entrance to the larynx.

Many modifications of O'Dwyer's tubes have been placed on the market. None of them possesses any advantage over the original model made by a faithful manufacturer, who worked under O'Dwyer's personal supervision, and most of the modifications are unfit for use.

The short tubes of Bayeux, which are so constructed that they may be pushed from the larynx by pressure on the trachea (enucleated), thus avoiding the use of the extractor, have obtained a certain vogue in France and other European countries, but have not been adopted in this country.

**Indications for Intubation.**—A patient with laryngeal diphtheria, having been given antitoxin, however great the probability that the operation will be ultimately required, should never be intubated until absolutely necessary. When, however, there is cyanosis, difficult breathing, marked retraction about the epigastrium and clavicles, and auscultatory

FIG. 91



Intubation in reclining position. First stage of the operation.

evidence that the air is not entering freely the bases of the lungs; when the pulse is weak and irregular, the patient restless and evidently being worn out, the operation should no longer be delayed.

**Method of Performing Intubation.**—The operation may be performed with the patient in the upright position or reclining; the latter possesses the advantage of absolutely requiring but one assistant, and even that one may be dispensed with in an emergency. It is less frequently employed than the upright position. In either case the patient should be pinned tightly from the level of the shoulders to the feet in a sheet or blanket, the arms being confined to the sides (Fig. 91). The

chest should be left bare. If the operation is to be performed in the upright position, the nurse or assistant, sitting upright in a straight-backed chair, holds the patient against the left breast by crossing the arms in front of the body. The legs of the patient are gripped between the nurse's knees. The second assistant grasps the patient's head firmly between her hands, the thumbs on the occiput, the little finger, and, perhaps, the fourth finger, being placed under the ramus of the jaw to pull it upward so that the neck is slightly extended (Fig. 90).

When intubating in the reclining position the patient is laid on its back upon a table, the head extending beyond it. The assistant should hold the head in the way just indicated for the upright position. It is sometimes advisable for a second assistant to keep the child from moving about by holding the lower extremities. The proper tube having been selected and tested to see that it slips readily from the obturator, and its eye is threaded, preferably by a strand of braided silk, the mouth gag is introduced between the back teeth on the left side and opened widely, the handles of the gag being included between the left hand of the nurse and the patient's cheek (Fig. 91). In younger children without back teeth, it is well to pad the jaws of the gag or dispense with it altogether in order to avoid wounding the gums.

The operator then inserts his left index finger into the patient's mouth, finds the epiglottis and drags it directly forward; at the same time crowding his finger as much as possible to the left he passes the tube directly in the middle line, and hugging the tongue as closely as possible under the edge of the finger-tip until the tube engages in the rim of the glottis. At the beginning of the operation, whether the patient is upright or reclining, the handle of the introducer should be parallel to the body of the child. As the tube approaches the glottis the handle is gradually raised until, as it engages between the vocal cords, the introducer passes beyond the perpendicular to the child's body. The tube then pointing directly down the trachea, is passed gently between the vocal cords to a distance of about two-thirds of its length, when the left forefinger should be removed from the epiglottis and placed on the side of the head of the tube, pushing the latter into place and holding it there at the same time that the obturator is released by means of the right thumb and withdrawn quickly by a slightly lifting and rotary movement from the mouth. The gag is then removed. If the tube is properly placed in the trachea there will be a succession of hollow coughs, together with the expulsion of more or less mucus and blood. The symptoms of stenosis are immediately relieved, cyanosis disappears, and the child, already exhausted, lies contented and very often sleeps. The cord should be left in for a few minutes so that the tube may be removed quickly in case of possible obstruction. In young children, and in older ones, if so desired, it may be left indefinitely, but under ordinary circumstances it is better to remove it, as it is often a source of annoyance and is frequently bitten off. For its removal it is only necessary to cut one end at the corner of the mouth, and, without inserting the gag, quickly place the left forefinger on the tube and pull

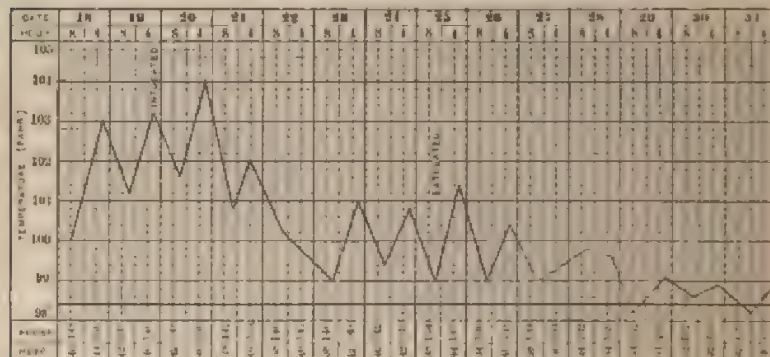


it out. If the string is to be retained it should be fastened by means of a strip of adhesive plaster to the left cheek.

In order to perform the operation successfully the following points should be emphasized: See that the child is held properly and is immovable, and that the proper tube has been selected and slips freely from the obturator when released. The introducers should be held between the thumb and fingers and never grasped firmly, the tube should be introduced as nearly as possible in the middle line, and finally the left forefinger should never leave the epiglottis until the tube has been well introduced into the larynx. In difficult cases several short efforts at introduction are far preferable to prolonged attempts.

**Extubation.**—In this operation the position of the patient is exactly the same as that required for intubation: the left forefinger is either placed on the epiglottis, serving as a guide for the beak of the extractor, or preferably on the arytenoid cartilages, the beak of the extractor being then passed along the middle line of the pulp of the finger, and by the quick elevation of the handle it glides into the opening of the tube. In order to avoid laceration of the tissues by premature opening of the jaws of the extractor, the thumb should not be put on the spring until the closed beak is well within the larynx. The spring is then pressed on firmly and continuously, the tube removed from the larynx by a combined lifting and rotary movement (Fig. 91).

FIG. 92



Temperature chart. Intubation and extubation in laryngeal diphtheria.

**WHEN TO EXTUBATE.** The time during which it is necessary to leave the tube in position depends on the severity of the case, the general condition of the patient, and the evidences of toxemia. The younger the child, the longer the time required for the retention of the tube. For children over two years of age it is usually customary to remove the tube on the third day. In many cases reintubation will be necessary, though with the use of antitoxin this is much less often necessary than formerly. When there is evidence of obstructed breathing with the tube in place it should always be removed immediately, as it not infre-



quently happens that its lumen is blocked by thick mucus or membrane, although under these circumstances autoextubation usually occurs.

**DIFFICULTIES OF THE OPERATION.**—No physician should undertake to perform this operation without thorough training on the cadaver. In unskillful hands the child may be killed by prolonged attempts to introduce the tube. The soft parts may be lacerated, with subsequent infection and false passages made, especially through the ventricles of the larynx.

It occasionally happens that membrane is pushed down before the entering tube, in consequence of which the symptoms of stenosis instead of being relieved are increased, the patient presenting all the symptoms of sudden asphyxia. The tube should be immediately withdrawn, the loose membrane usually being promptly expelled; after which the tube may be reintroduced. That this accident is not common is shown by the fact that it has seldom or never occurred in a long series of intubations performed by skilled operators, and there can be no doubt that this condition has often been held responsible for the accidents due to lack of skill above enumerated. Subglottic stenosis, or edema of the glottis, occasionally causes some difficulty in introducing the tube, and is the only occasion in which a certain degree of force is necessary in performing the operation.

**RETAINED TUBE.**—Frequent reintubations are due to ulcerations within the larynx, with more or less destruction of the cartilage, cicatrices, granulations and paralysis of the intrinsic muscle. These conditions much more frequently follow cases of mixed infection, the use of improperly constructed tubes and unskillfully performed operations.

**FEEDING OF INTUBATED CASES.**—The method of feeding by gavage has already been described (p. 408). In infants and young children this may be kept up throughout the period of intubation.

Cassellberry's position may be employed with the child lying across the nurse's lap, the head slightly back and feet elevated on a chair, and the food given with a spoon or from a bottle. With older children very little difficulty is usually experienced in feeding after the first day or two. They are quickly taught to take their food in a natural position and without exciting more than an occasional mild attack of coughing. The food should be given slowly in small quantities, semisolids often being more readily managed than liquids.

### TRACHEOTOMY.

For the performance of this operation an anesthetic should be given when possible. The patient should be placed on a table under a good light, the head well back and steadied by an assistant; the index finger of the left hand should be used to locate the cricoid cartilage, the larynx held firmly in the median line by the thumb and remaining fingers. An incision is then made exactly in the middle line from the cricoid downward for a distance of about 3 cm., the skin and subcutaneous tissues

being divided. The left forefinger is then placed over the bare trachea at the upper angle of the wound, and by means of a bistoury an incision made large enough to admit the finger-tip, after which the canula is introduced in the tracheal opening, the finger being withdrawn. A tracheal dilator may be employed instead of the finger. When cannula breathing is established the tube is fastened in place by means of tape about the neck, a strip of antiseptic gauze placed about the wound and over the opening of the canula. It is unnecessary to state that every antiseptic precaution should be taken before performing the operation except in emergency cases (Fig. 93).

FIG. 93



Showing correct position for performing tracheotomy.

Among the accidents which may happen during and after the operation are difficulty in introducing the canula on account of a too small tracheal incision, or because the trachea has not been opened, a false passage being made with the canula. Hemorrhage at the time of the operation is usually not severe unless the incision has been carried too low. Secondary hemorrhage occasionally occurs.

**Effects of the Operation.** The immediate effects of introducing the canula are exactly similar to those following intubation. There is, however, more apt to be a rise in temperature lasting for a day or two.

**Complications.** Infection of the tracheal wound is apt to follow emergency operations and in those in which proper antiseptic pre-

cautions have not been taken. Diphtheria of the wound occasionally occurs and sometimes erysipelas. Extensive suppuration with sloughing of the tissues and occasionally gangrene are sometimes seen. As in intubated cases the most frequent and dreaded complication is that of bronchopneumonia.

**Treatment of Tracheotomized Cases.**—The internal canula should be removed and cleaned at first at intervals of two or three hours, and always when there is sign of obstruction. The external canula should be removed every day and thoroughly cleaned, together with the wound. After the second or third day, if there are no further signs of laryngeal obstruction, the canula may be removed permanently and the wound carefully dressed. Healing usually takes place rapidly. To prevent the recurrence of laryngeal stenosis the same general treatment is indicated as that advised for intubated cases.

**Indications for Tracheotomy.**—Tracheotomy is not the operation of election in this country. It should be performed when for any reason intubation is not possible, and as a secondary operation when, intubation having failed to give relief, there is reason to suppose that the obstruction is due to tracheal membrane. Lastly, in some cases autoextubation occurs so constantly that larger and larger tubes have to be used in rapid succession and the integrity of the larynx is endangered. Here, again, tracheotomy may be resorted to. It may be mentioned that at the New York Foundling Hospital with a very large number of laryngeal cases, occurring both before and since the introduction of antitoxin, tracheotomy has never been called for or performed, possibly due to the fact that intubation has been performed by members of the staff who have been thoroughly trained in the operation. At other hospitals, where the patients very often have been intubated before admission, and with variable skill, secondary tracheotomy is not infrequently required.

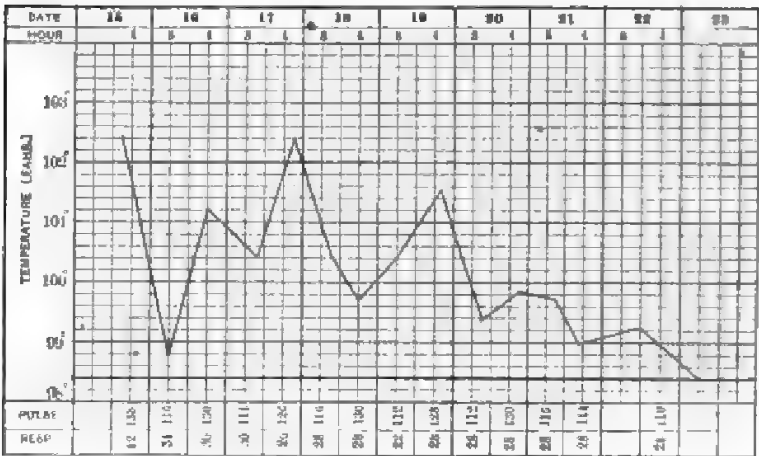
### PSEUDODIPHTHERIA.

Pseudomembranous inflammations due to other than the diphtheria bacilli, generally the pyogenic cocci, occur in by far the greater number of cases in scarlet fever, measles, and, less often, influenza and exanthemata other than those named. They may be regarded as a local evidence of the mixed infections so likely to take place in these diseases. Their presence adds to the danger of the primary disease in that they render probable the occurrence of local suppurative conditions and in their worst form of general sepsis. The symptoms of this condition are masked by those of the primary disease.

Primary pseudodiphtheria is not an uncommon condition, especially in institutions. In the mild and most frequent form the membrane is usually confined to the tonsils, frequently in the form of a small, grayish-white patch sunken beneath the surface of the tonsil. The latter is red and swollen. There is a rapid and marked rise in temperature and in the pulse rate and the patient very often feels acutely sick. The cervical

lymph nodes are more or less involved. The attack lasts a number of days and, as a rule, is not followed by complications, although suppuration of the nodes and middle-ear disease may occur. In the severe cases the membrane spreads rapidly and is apt to involve the pharynx and nose. There may be sloughing of the tissues of the throat, with a foul discharge from mouth and nostrils, great swelling of the lymph nodes, very often followed by suppuration. Middle-ear disease is very frequent, bronchopneumonia and general sepsis are apt to occur and terminate the disease fatally. In the severe cases the larynx also may be involved with symptoms of laryngeal stenosis not to be distinguished from that of true diphtheria. This occurrence, however, is very rare, and even

FIG. 94



Temperature chart. Case of pseudodiphtheria, with recovery.

if the larynx be involved it is doubtful if in such cases a true laryngeal membrane is present. Cases of primary streptococcus croup are occasionally described. The diagnosis of such a condition is based upon the fact that one or more cultures taken from the throat fail to show the presence of Klebs-Loeffler bacillus, but, as already pointed out, this is a not infrequent occurrence in cases which subsequently show the diphtheria bacilli in great numbers at a later stage of the disease. In my opinion such cases should always be looked upon with suspicion, and even if their occasional occurrence be admitted, they are excessively rare. The symptoms are those that have been described and are exactly similar to cases of true laryngeal diphtheria and need not here be dwelt upon (Fig. 94).



## CHAPTER XVII.

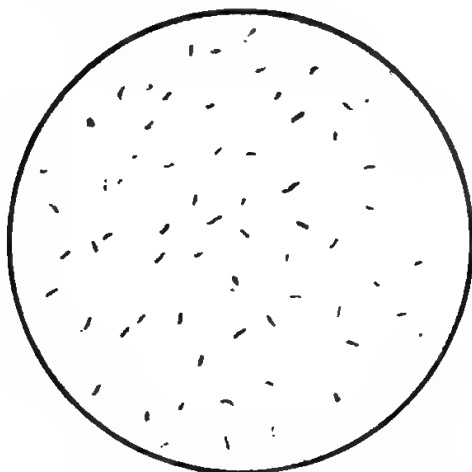
### TYPHOID FEVER—MALARIA—EPIDEMIC CEREBROSPINAL MENINGITIS—INFLUENZA.

#### TYPHOID FEVER.

By ISAAC A. ABT, M.D.

**Etiology.**—Typhoid fever is due to the presence in the body of the typhoid bacillus. This organism, so far as is known at the present time, is pathogenic only for man. The bacillus thrives best at the temperature of the human body, but will grow to some extent at lower temperatures. It is readily killed by heating to 70° C. (150° F.) and by antiseptics (Figs. 95 and 96).

FIG. 95



Typhoid fever bacilli. One-twelfth oil-immersion lens.

Most frequently typhoid fever is contracted by infected drinking-water, and next by infected milk. In cities where the water supply is contaminated by sewage the disease is always prevalent. Where the source of water is kept free from pollution, typhoid becomes comparatively rare. In certain cities of Europe in which especial precautions have been taken to obtain a pure water supply, typhoid fever is practically unknown.

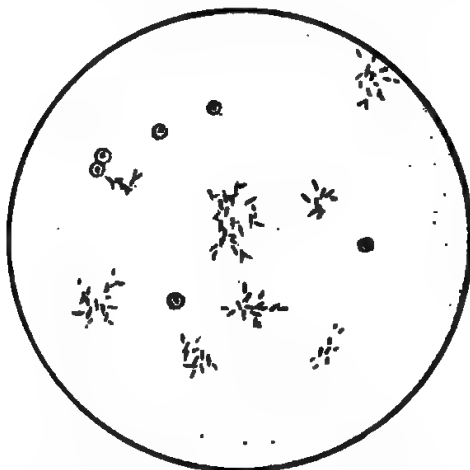
Milk may be infected by being adulterated with water which contains typhoid bacilli, or by washing cans with contaminated water. Wells in

the country are frequently infected from cases of typhoid fever in the dairyman's family. The udders of cows, too, may be washed with infected water; or contaminated dust gains access to the milk.

Flies carry infection in two ways: (1) fecal matter containing the typhoid germs may adhere to the fly's legs, wings, or body and be mechanically transported, or (2) the bacilli may be carried in the digestive organs of the fly and deposited with its excrement. This latter method has not been proved. There can be little doubt, however, that the fly may carry germs from infected excreta to foodstuffs.

The specific germ not only grows in milk, but also in milk products. It will live in butter for many days, and in cheese for a short time. The oyster can harbor and carry the typhoid bacillus. Infection of the oyster usually takes place during the time when it is being freshened

FIG. 96



Positive Widal reaction, showing agglutination of typhoid fever bacilli—with blood from typhoid patient—at end of thirty minutes.

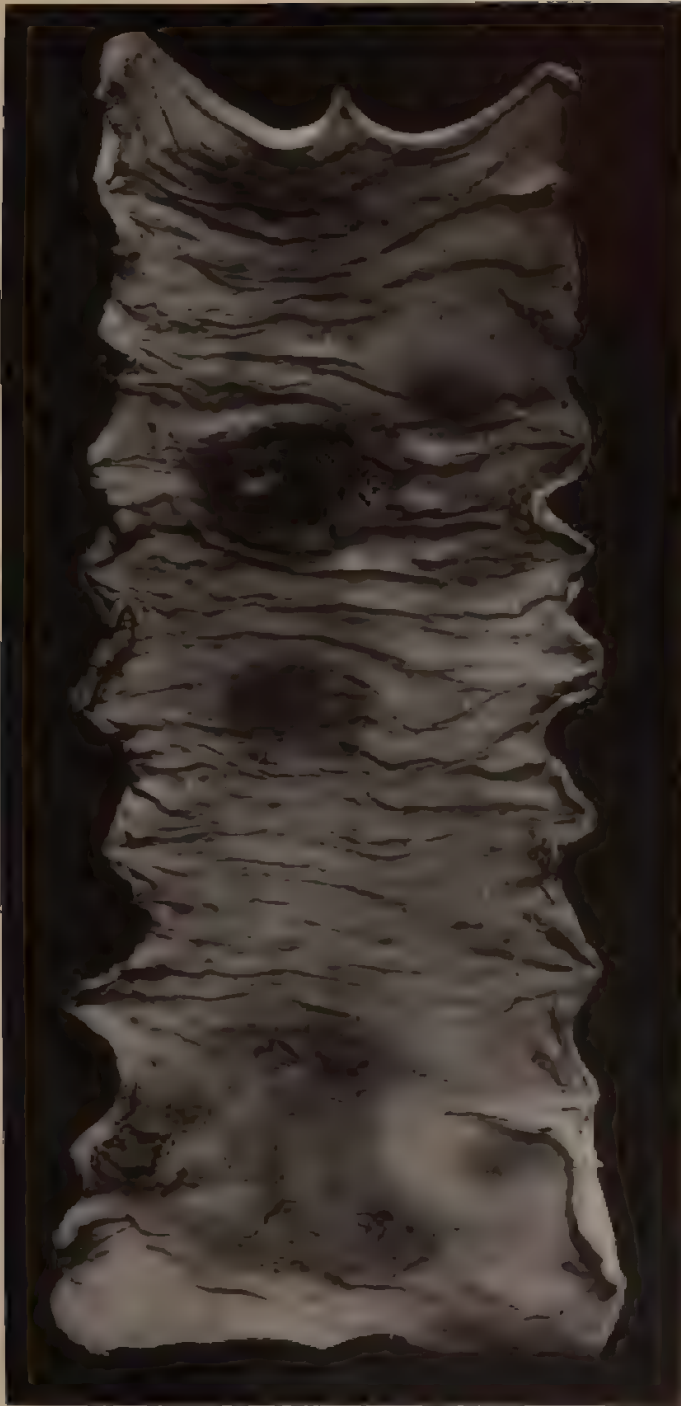
in water that is contaminated by sewage. Other foods may become infected, particularly those which are eaten raw and previously washed in infected water.

*Predisposing Factors.*—Sex appears to have no influence on the occurrence of the disease.

*Season.*—Typhoid fever is endemic in most localities. It occurs all seasons of the year, although it is commonly stated that it is more prevalent in the fall months. Osler says typhoid is essentially an autumnal fever, and more than one-half of his cases were admitted in August, September, and October.

*Frequency in Children.*—From 200 cases of my own which were observed for the most part in the Michael Reese Hospital, Chicago—the following tabulation of ages is made:

PLATE XI.



Typhoid Ulceration of the Ileum.

(From the Laboratory of the College of Physicians and Surgeons, Columbia University, New York. Photograph by Dr. Edward Leaming.)





0 to 1 year . . . . .	2	6 years . . . . .	14
1 to 2 years . . . . .	2	7 " . . . . .	12
2 " . . . . .	7	8 to 14 " . . . . .	129
4 " . . . . .	18		
5 " . . . . .	16		200

**Pathology.**—The opinion is general that typhoid fever in infants produces less pronounced anatomical changes than in older children and adults. In some cases, however, autopsies on comparatively young children have shown anatomical changes not dissimilar to those which occur in adults. As a result of the typhoid infection, hyperplastic processes in the intestine are more pronounced than the ulcerative ones. The typhoid bacillus in the small intestine produces swelling of the solitary follicles and Peyer's patches. These appear raised, of a rose-red color, and surrounded by a circumscribed area of redness. The process does not remain localized in the intestine. Recent investigations, particularly the bacteriologic studies of the blood, have shown that in the vast majority of cases, typhoid bacilli are found in the circulating blood before the end of the first week of the disease. This explains the presence of typhoid lesions in every part of the body. Mallory believes that the primary effect of the localization of the typhoid bacillus is a multiplication of the endothelial cells lining the lymph channels in the intestine. In these structures he has found mitotic figures and a marked increase in the endothelium, with the lumen of the blood and lymph vessels entirely obliterated. The blocking of the bloodvessels results in stasis; very soon fibrin is precipitated and thrombosis results. The poor nutrition consequent upon this process leads to necrosis. It should again be emphasized that in infants and very young children ulceration of the solitary follicles and Peyer's patches is the exception; in older children and adults it is the rule. (See Plate XI.)

When ulceration occurs it is most abundant in the lower half of the ileum, but the ulcers may occur in the large intestine as well. The lymph follicles swell in the early stages of the disease; they are sharply outlined and flat, at first deep red, then grayish-red in color; if necrosis occurs the patches become grayish-yellow, and the central portion or the whole of the swollen patch becomes necrotic. The necrotic portion is cast off and a clean ulcer with sharply defined, raised edges appears in its place. In some of the patches no ulceration takes place. Many of the solitary follicles may ulcerate. In some cases almost the whole circumference of the bowel loses its mucous membrane. Stenosis of the bowel sometimes results from contraction of the connective tissue. After the ulcers have healed a characteristic granular blood pigmentation is observed. This is referred to as the "shaven beard" appearance. Peyer's patches which present a slightly depressed scar, readily indicate a past typhoid. As a rule, the process of ulceration is confined to the mucosa and submucosa, though the muscular coat may be involved. This may result in severe hemorrhage from branches of the mesenteric vessels; or the serous coat may be involved in the ulcerative process and

perforation into the abdominal cavity results. In such a case diffuse peritonitis is inevitable.

Characteristic features of the lesions are:

1. The ulcers are longest in the long axis of the gut, thus distinguishing them from tuberculous ulcers, which are usually longest in the transverse axis. The longitudinal direction of the typhoid ulcers is explained by the fact that the degenerative process is confined altogether to the lymph follicles. In tuberculosis the ulcer tends to spread in the direction of the lymphatics which run in a transverse direction.

2. The peritoneum remains free from exudates; the peritoneal surface is usually smooth and glistening, no matter how closely the ulcer approaches it. Adhesions between the various loops of intestines, such as frequently observed before the rupture of an appendix, rarely occur in typhoid. This explains the diffuse peritonitis which results after intestinal perforation.

*In the mesenteric lymph nodes* the endothelial cells proliferate enormously. The nodes increase in size, and a cut-section shows areas of necrosis. When the node is cut through, its substance bulges out, giving a convex appearance. This indicates the swelling and existent pressure within the node. Suppuration sometimes occurs. Rupture of the node has been observed. Mesenteric nodes are always more or less enlarged; in consistency they are soft; their color is white or pink and the necrotic areas appear yellowish.

*Spleen.*—It is usually three or four times its normal size. The capsule is distended. The organ is usually soft and at times the Malpighian follicles are prominent and white or gray in color. Their enlargement may be ascribed to lymphatic hyperplasia similar to that which occurs in the lymph follicles of the intestine. The spleen is largest at the height of the disease and at that time dark and congested, sometimes almost fluid.

*Mucous Membranes.*—Various mucous membranes may be involved. Bronchitis and laryngitis occur. The cricoid and arytenoid cartilages may become secondarily involved, and stenosis of the larynx and aphonia result. Death may occur as a result of laryngeal ulceration.

*Serous Membranes.*—The serous membranes are not commonly affected. Pleurisy is rare. Peritonitis may occur in consequence of perforation of an intestinal ulcer, or rupture of a suppurating, mesenteric lymph node. More rarely, peritonitis is caused by the migration of typhoid bacilli through an intact serosa. It seems more plausible, in the light of our present knowledge, to consider this a hematogenous infection.

Abscesses do occasionally appear in every part of the body on account of the wide distribution of the typhoid bacillus, and of a secondary infection with pyogenic organisms. Suppurative processes occur in the skin, bones, or joints. Brain abscesses have been reported, though only one case is recorded in which a pure culture of the typhoid bacillus has been found.

*Visceral Changes.*—Parenchymatous or fatty degenerations may be found in any or all of the tissues. The liver and kidneys are swollen, and their markings become indistinct. In the kidney the inflammatory

changes range from a mild, cloudy swelling to a well-marked nephritis. Pyelonephritis and abscess formation rarely occur.

It is assumed that the gall-bladder is infected with typhoid bacilli in nearly every case, and may lead to the formation of gallstones at a later period of life. Suppurative cholecystitis is known to occur and the inflammation extend into the small bile-ducts. In one of my cases a diffuse cholangitis was observed.

Bronchopneumonia is frequently found as a terminal lesion in fatal cases, though it is sometimes secondary to a diffuse bronchitis, and may be considered the immediate cause of death. Lobar pneumonia occurs in a few cases; occasionally due directly to the action of the typhoid bacillus.

The heart muscle usually shows a mild grade of parenchymatous degeneration. In the severe cases the myocardial changes may be more extensive and partake of the interstitial type. After recovery, the effect of the myocardial inflammation usually disappears, though the heart muscle may remain permanently damaged. Owing to an enfeebled heart action, thrombi may form in the auricles and be swept on into the general circulation. In this way infarcts occur in the spleen, the kidneys, and lungs. Hemiplegias have been observed as the result of typhoid fever. In two cases in which autopsies were held thrombosis of the middle cerebral artery was found.

**FETAL TYPHOID.**—Pregnant women suffering from typhoid abort in about one-half of the cases, and the fetus is born dead (Klautsch). The causes advanced are: (1) high temperature; (2) the accumulation of toxins in the maternal blood; (3) death of the fetus. It has been found experimentally that the intravenous injection of typhoid cultures into pregnant rabbits and guinea-pigs resulted in abortion (Frascome).

Intrauterine typhoid is from the first a general septicemia. Bacteriologic examinations give corroborative evidence of the presence of the typhoid bacilli in the blood of the fetus. They have been found in the spleen, in the heart's blood, and the liver. The septicemic nature of the infection accounts for the extreme mortality in fetal and congenital typhoid. For this reason, and possibly also because the intestines are not functioning, the classical intestinal lesions of typhoid are absent in infants. The fetus usually dies *in utero*. It may be born alive, but feeble and suffering from the infection, in which case death occurs within a few days without definite symptoms. It is possible for the fetus to sicken, recover from its infection, and be born alive and well. Infection does not always occur. A pregnant woman does not necessarily transmit the disease to the fetus.

Newborn or young infants whose mothers are suffering from typhoid fever may exhibit the Widal reaction without other symptoms. In such cases it is possible that the infants have had typhoid fever *in utero*, or that the agglutinating power may have passed from the diseased mother into the healthy child through the placenta or the mother's milk.

**INFANTILE TYPHOID.**—Typhoid fever occurs more rarely in infants than in older children or adults. All the statistics since the intro-

duction of the Widal reaction and the other more accurate means of diagnosis show that typhoid fever in children under two years of age is not of frequent occurrence. Infants are less exposed than older children, though they are not known to possess immunity to the infection. From what has already been said, it would seem that the infant is susceptible to typhoid infection, since it may become infected *in utero* through the placenta.

It appears from a study of typhoid fever in infants that the symptoms are essentially the same as in adults; but the course is shorter and the mortality higher. These conclusions must be accepted as essentially correct, if the cases on which they are based are typical. It is possible, however, that they comprise only the severe varieties, and that many milder cases have been mistaken for other infections.

The autopsies on infants show the absence or slight degree of intestinal involvement in fatal cases. In this way they differ from the fatal cases seen in adults. The enlargement of the mesenteric nodes is moderate, although the spleen is almost always considerably enlarged. In sharp contrast to the mildness of the pathologic changes is the severe general infection during life, with its great mortality. In this it resembles fetal more than adult typhoid. These conditions as they occur in fetal typhoid are traceable to the blood infection. This also obtains, though to a lesser degree in infantile typhoid, and explains the wide disproportion between the pathologic changes and the severity and fatality of the disease.

*The Course of the Disease.*—In a general way the cases among infants may be classified as mild and severe. The following are illustrative:

*Mild Type.*—A male baby, aged twenty months, well developed and of healthy parents, had occasionally been ill with mild gastroenteric derangements which had previously yielded readily to treatment. After the present illness had persisted for a week the mother sought medical aid. The temperature was fairly constant, ranging between 101° and 103° F. The mother took occasion to say that she did not believe the child to be suffering from one of his usual gastrointestinal infections, because the stools showed a more perfect digestion and were less foul-smelling than during his previous intestinal attacks. The child was pale; there was an enlarged spleen; his muscles were somewhat flabby; he was restless. He objected to restraint and did not impress one as being severely ill. The Widal examination in a few days was positive and leukopenia was indicated by a leukocyte count of 4800. A few roseolar spots were observed on the abdomen. The fever continued in all about sixteen days and recovery was uneventful (Fig. 97).

*Severe Type.*—One sometimes sees very severe cases in young children where the prostration is extreme and the fever high. These children are not inclined to play but prefer to lie in their cribs or in the mother's lap undisturbed. Such a case I saw in an infant about twenty-one months old. He appeared greatly prostrated; fever was high; the pulse and respirations were accelerated; spleen was large; the abdomen tympan-



itic, and the trunk, both anteriorly and posteriorly, was dotted with well-marked rose spots. He recovered after having a continued fever for four weeks.

**Symptomatology. Prodromata and Mode of Onset.** The symptoms preceding the actual onset of the disease are very difficult to elicit in infants and young children. It is not uncommon that the disease is ushered in abruptly and marked by sudden rise of temperature and vomiting. A sudden rise of temperature was recorded in all the young children under my care; vomiting occurred nineteen times at the very onset of the disease. Convulsions are said to occur rarely. In one of my cases the disease showed its beginning by a convulsive seizure. Chill or chilliness was frequently complained of by the older children at the beginning, and headache was more frequently noted than any

FIG. 97

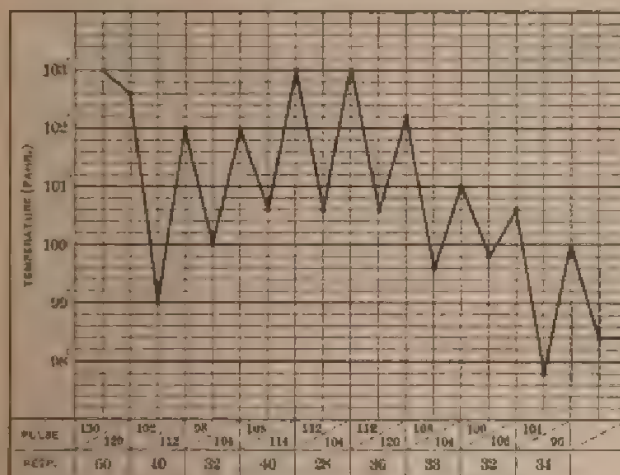


Chart of a mild case of typhoid fever.

other single symptom except fever. Epistaxis, which is of common occurrence in adults, seems to be less frequent in children. Out of 200 cases it occurred only four times during the early days of the disease. Delirium was observed once; abdominal and muscular pain was present thirteen times; anorexia, prostration, nausea, pharyngitis, and insomnia were occasionally noted as prodromal or initial symptoms of the disease.

Some cases are ushered in by pronounced nervous manifestations in the form of meningeal symptoms. I have observed several such cases in which it was impossible during the first few days to exclude the diagnosis of meningitis. A three-year-old boy was taken suddenly ill with high fever. His temperature was 104° F., he had excruciating headache and vomited frequently, and very soon passed into a comatose condition. The neck was rigid; the pulse irregular; the pupils

were symmetrical, though there was a slight deviation of the eyes; the reflexes were increased; ankle clonus was present; Kernig's sign was marked. The Widal was positive on the second day of observation. On the fifth day the meningeal symptoms had entirely disappeared and the case ran the course of a typical typhoid.

*Fever.*—The temperature course is not altogether typical; in older children it sometimes rises gradually during the first week of the disease. By the end of the first week a maximal point in the fever is reached; in which case, after touching  $104^{\circ}$  or  $105^{\circ}$  F., the curve becomes remittent. The remissions average about  $1.5^{\circ}$  after the disease is fully established. With the advent of the second week the remissions continue, though the general temperature range may be lower. On the other hand intermissions may occur. In the severer cases hyperpyrexia may continue for a long time—six to eight weeks is not uncommon—or the disease may be still further protracted by relapses after the temperature has fallen to normal or nearly normal.

Among infants and young children the temperature curve is less regular than above described. The initial rise is sometimes rapid. During the acme of the disease the fever often remains high, with little variation between the morning and the evening temperatures. During the second week the remittent character of the fever is less marked than in adults. According to Morse's figures it is absent altogether in about one-half the cases. The range of temperature is usually higher, though less significant of gravity, than in cases of like severity among adults.

The course of the fever is modified by complications, the occurrence of pneumonia, furuncles, or otitis causing an increase in the height or an irregularity of the fever curve. Excitement frequently influences the temperature curve. In the hospital wards it is commonly observed that all the typhoid fever children exhibit a slightly higher temperature on visiting days when parents and friends are admitted.

Fever sometimes persists after the disease has apparently run its course, or after the temperature has once come to normal. These post-typhoidal temperatures do not necessarily signify a relapse; they may be due to complications, to intestinal toxemias, or occasionally to inanition.

*Hypothermia.*—The fever may fall below normal after a tub bath; this is particularly observed during the third or fourth week of the disease. After hemorrhage the temperature may drop from  $103^{\circ}$  F. to  $98^{\circ}$  or  $95^{\circ}$  F. In these cases the fever may not rise for twenty-four hours or longer, when it again ascends to the high point. During convalescence the temperature may be subnormal— $96.5^{\circ}$  or  $97^{\circ}$  F. for days in succession. This is particularly noted in protracted cases with great emaciation.

Relapses occurred in 10 per cent. of my cases. Usually they are mild and run a short course, though sometimes they are severe and exceed the primary attack in intensity and duration. In one of my cases a second relapse occurred, that is, a third attack of the disease.

The last relapse was preceded by twenty days of normal temperature, and the patient had apparently recovered. During the relapse the spleen usually becomes enlarged, the roseola reappear, and are the same as in the primary attack. Recovery occurred in all the cases of relapse which came under my observation.

*Pulse.*—In infants and young children the pulse rate frequently reaches 150 to 180, which, in older patients, would certainly indicate a fatal termination. In older children it follows the type of adults and is relatively slow. A dicrotic pulse occurs only in a small number of cases. Intermittence is sometimes noted. Bradycardia is occasionally observed during the febrile period. In one of my cases, that of an older child, the pulse varied between 50 and 70 per minute during the entire febrile period.

*Heart.*—It is not uncommon to hear systolic murmurs over the base of the heart. Many of these murmurs disappear as the fever subsides. In some of the cases an old valvular lesion, which has antedated the typhoid fever, explains the murmur. In a few cases it is due to myocarditis. A boy, ten years old, who had suffered a severe attack of typhoid with very high fever had a striking irregularity of the pulse, which began during the second week of the disease and continued during the stage of defervescence and convalescence. During the latter period the heart's action was decidedly irregular. For example, at 11 o'clock of one day his pulse was 76; one hour later, without any apparent cause, it was 136. This condition became more marked as convalescence advanced, so that at one time the heart's action was 50 or 60 per minute, while at another hour it was 100 or 120 per minute. After prolonged convalescence the heart's action became regular and the patient recovered.

*Spleen.*—The spleen is usually enlarged, but this is not always discerned during the first days of the disease; at the end of the first week it can, in most cases, be palpated. In many cases it continues swollen after the fever has abated; if the spleen becomes normal in size it again becomes palpable if a relapse occurs. The enlargement of the spleen is elicited more easily by palpation than by percussion. Deep pressure is not necessary. Tenderness upon palpation is seldom observed. A relapse may take place without splenic enlargement.

*The Gastroenteric Tract. Tongue.*—During the first few days the tongue may show no changes. Later, when the temperature is high, it becomes coated and dry; the dorsum is white, the tip and edges red, or it may be fissured in the longitudinal direction and covered with a dry, brownish fur. As the disease progresses the epithelium is desquamated and the papillae stand out prominently. In some cases the tongue is intensely red; in others the papillae are prominent, resembling the strawberry tongue of scarlet fever.

*Mouth.*—At the height of the disease the mouth becomes dry, and, notwithstanding the most careful nursing, infection may occur. Catarrhal stomatitis and gingivitis are not uncommon, producing great distress. An increased flow of saliva is sometimes observed.



*Herpes labialis* is thought to be very infrequent, although recently its occurrence has been confirmed by numerous reports in the literature. It occurred in four of my cases.

*The Intestines. Tympanites.*—The abdomen may become distended, though not as a rule before the second week of the disease. In some of the severe cases the tympany is extreme and may be accompanied by diarrhea; or the tympany may be caused by constipation or impaction of feces. If severe, it may cause respiratory embarrassment.

*Stools.*—Constipation is more frequent than diarrhea. In some cases diarrhea may be present during the first week and constipation through the remainder of the disease. The diarrhea usually is slight, rarely it is severe in character. In the constipated cases no stool occurs unless an enema or suppository is given. The bowels may move once or twice a day without assistance. In younger children the stools are thin, frequently contain undigested milk particles, and sometimes mucus. They may be normal in color, and vary in consistency slightly from the normal. Fecal impaction occurs at times. In two of my cases the impaction occurred during the period of convalescence before the patients had been fed on solid food. In both a slight temperature, marked tympany, and a feeling of fulness and distress in the rectum occurred. After mechanically removing a large quantity of the feces the symptoms subsided.

*Hemorrhage.*—In general it may be said that hemorrhage is not as frequent in children as in adults. It is more often observed at the end of the second week than at any other period of the disease. I observed hemorrhage five times in 90 cases. In 2 it was slight; in 1 moderately severe; in a second case two severe hemorrhages took place; in another repeated attacks of bleeding occurred from the bowel; the quantity of blood lost each time was small. With each hemorrhage the temperature fell from 103° to 99° or 100° F.; the pulse became rapid, the skin blanched; the patient complained of abdominal pain before the attacks.

The ages of patients in whom hemorrhages occurred were as follows:

6 years old	. . . . .	Eighth day of relapse.
10 " "	. . . . .	Eleventh " disease.
12 " "	. . . . .	Seventh " "
10 " "	. . . . .	Sixth " "
11 " "	. . . . .	Eighth " "

Sometimes stools containing a small quantity of blood and mucus are observed during defervescence, or at the beginning of convalescence, and indicate the presence of an ileocolitis in contradistinction to a bleeding typhoid ulcer.

*Perforation.* It would appear from the literature that perforation is not common. Several cases, however, have come under my observation.

There is no regularity in the symptoms, and no symptoms or group of symptoms are pathognomonic for perforation. Osler emphasizes the following points: Sudden abdominal pain and alterations in the



**pulse** and respiration. The physician should never disregard such **warnings**, but see the patient immediately. Those handling typhoid fever patients must remember that perforation comes in many guises, and that to detect its presence from any constant combination of signs is **hopeless**. There are, however, certain points which stand out, and of these a most important one is the onset. This is sudden and usually **with** pain. In the cases which have had pain for days before the perforation there may be an exacerbation which attracts attention, or there may be nothing more striking than before. After the onset, Osler says the most important constant features are pain, often in severe paroxysms, tenderness, and some rigidity. Other symptoms may be present, such as vomiting, sweating, change in the temperature, pulse, and respiration, impairment of abdominal respiratory movements, muscle spasm, decrease in the area of liver dulness (which is one of value if one has careful notes of its extent previously, and is of little importance where there is marked abdominal distention), movable dulness in the flanks, and leukocytosis. The final interpretation to be given to leukocytosis is far from being made. Cushing and Finney show in some of their cases a fall and not an increase in the leukocytes. The data are: 1. The appearance of leukocytosis in the course of typhoid fever points toward some inflammatory complications in its early stage. 2. If this complication be a peritonitis and remain localized, associated possibly with the preperforative stage of ulceration, or with a circumscribed, slowly forming peritonitis after perforation, it may be, and usually is, **signalized** by an increase in the number of leukocytes. 3. If, however, a general septic peritonitis follows, the leukocytosis may be but transitory and overlooked, as it disappears concomitantly with the great outpouring of leukocytes into the general cavity.

**Pain**, tenderness, rigidity, change in the pulse, respiration, and temperature may all be found without perforation. The only other means of **making** a positive diagnosis is exploration.

McCrae, in the *Johns Hopkins Reports*, compares the operation for perforation with that for appendicitis. He remarks that while exploration has often been attempted too late, early operation has never been regretted, and adds that a positive diagnosis of perforation in every case before operation is not to be expected, nor is it wise that it should be demanded. What we have to decide is whether the condition is of sufficient gravity to warrant an exploratory operation to ascertain the existence or not of some abdominal complications.

**Pain on Abdominal Palpation**.—Older children sometimes complain of pain in the ileocecal region; this is found particularly in cases where extensive ulceration is present. In younger children it is obvious that this sign is valueless.

**Respiratory Organs**.—Bronchitis occurs very frequently, usually at the end of the first week. It is present in most of the severe cases, though it may appear in those of mild type. Bronchopneumonia and lobar pneumonia may occur; edema of the larynx is rare. Pleurisy, abscess, and gangrene of the lungs have been reported.

*Urine.*—The urine in the earlier stages of the disease is scanty, hyperacid, highly colored, with high specific gravity. The amount increases as soon as convalescence begins, the increase in some instances being very decided, so that polyuria is occasionally seen. The urine, which at first is dark, becomes lighter and almost colorless during the post-febrile period. The specific gravity varies also, depending upon the height of the fever, the quantity of liquid taken, the amount secreted, and the presence or absence of diarrhea. Urea is diminished during the early stages of the disease. Uric acid is increased; the chlorides are always diminished during the early stages. The toxicity of the urine is much increased; it is double that of normal urine, and is independent of the temperature; it remains increased during the entire course of the fever, and during convalescence. Albumin is frequently present. Hyaline and granular casts are observed in the nephritis of typhoid. Albumin was noted in 8 out of 90 cases, and hyaline and granular casts occurred in 13 of the 90.

The diazo reaction of Ehrlich is present in 90 per cent. to 95 per cent. of all cases of typhoid fever, and sometimes occurs before the Widal reaction can be obtained. Unfortunately for the diagnosis, it often occurs in other diseases which are readily confounded with typhoid fever, such as miliary tuberculosis and measles. To carry on the reaction two solutions are required: (1) A saturated solution of sulphanic acid in 1000 c.c. of water and 50 c.c. strong hydrochloric acid; (2) a 0.5 per cent. solution of sodium nitrite. The latter should be fresh, as it is soon oxidized to sodium nitrate. Four drops of the second solution are added to 10 c.c. of the first, and the whole shaken with 10 c.c. of urine. Ammonia is then floated on the surface. If the diazo reaction is present, a bright-red ring should appear where the fluids meet. On shaking the whole mixture becomes deep red in color and the foam, too, is red. All of these changes must occur in order that the reaction may be considered positive. The diazo reaction is usually present by the end of the first week, but may be delayed until late in the course of the disease.

*Kidney lesions* in typhoid fever are due to the inflammatory and degenerative changes produced by the typhoid bacillus itself, or by other micro-organisms which enter the urine through the diseased intestinal wall, or, possibly, in some cases, by bacteria which enter the bladder through the lower urinary passages. Bacteriologic examinations of the urine show the presence of typhoid bacilli. In about 30 per cent. of our cases the colon bacillus, staphylococcus pyogenes aureus and albus were found associated with typhoid bacilli in the urine. The urine of fifteen children, which was examined bacteriologically, showed the presence of the typhoid bacillus five times. In some cases the colon and typhoid bacilli were found in the same urine.

The bacilli may be found as early as the second week or as late as the fortieth or forty-fifth day. Other micro-organisms like staphylococcus pyogenes aureus and albus may obscure or cause the disappearance of the typhoid bacillus. In the cases that contain the typhoid

bacillus, pus was almost invariably present, and the pus explained in part at least presence of albumin in the urine.

*The Blood.*—There is a reduction in the number of red cells. This occurs shortly after the onset of the disease. The diminution increases gradually; probably the greatest reduction is noted about the end of defervescence. On the average it is estimated that the maximum loss of red blood corpuscles is about 1,000,000 per c.mm. Hemorrhage from the bowels causes a further diminution in red corpuscles.

The hemoglobin gradually diminishes with the diminution in the number of red corpuscles; returning to normal more slowly than the number of red cells.

The number of leukocytes is diminished in typhoid fever. The decrease is progressive, the corpuscles becoming less as the disease becomes more advanced, so that at the height of the disease about 4000 or 5000 leukocytes per c.mm. are noted. Sometimes the number may be much lower. If the leukocytes increase considerably, it may be assumed that some influence such as hemorrhage, perforation, or inflammatory complication is responsible. An increase above 10,000 is abnormal for an uncomplicated typhoid. Cold baths, however, may cause a transient increase, so that three or four times the usual number of leukocytes may be noted after the completion of the bath.<sup>1</sup>

The different varieties of leukocytes show variations from the normal percentage. At the height of the fever the small and large mononuclear cells are relatively increased; the greatest decrease is noted in the polymorphonuclear neutrophils. The decrease of these cells keeps pace with the increase in the large mononuclear forms. The percentage of eosinophilic cells is diminished throughout the course of the disease, though, as convalescence approaches, the eosinophiles increase to a point rather above the normal average. In those cases in which a mixed infection has occurred as a complication an increase in the number of leukocytes is the rule, but if the patient be in a condition of prostration the leukocytes may not only fail to show any increase, but they may show a diminution in number. In a complication as severe as perforation, a complete absence of leukocytosis or a diminution in the number may be observed.

*The Skin.*—The most important change is the appearance of the roseolæ. They occur from the sixth to the tenth day, have been noted as late as the third and fourth week. They consist of rose-red papules which vary from 2 to 4 mm. in size. These are slightly raised and disappear on pressure. They tend to appear in successive crops; sometimes only a few appear, and at other times they are very abundant; they occur most commonly on the abdomen, also on the thorax, on the buttocks; sometimes they may be seen in the axilla and on the flexor surface of the arms. In one of my cases the roseolæ became the seat of purpuric spots, which persisted for several days, when they disappeared, leaving brownish stains.

<sup>1</sup>Thayer, Observations on the Blood in Typhoid Fever, Johns Hopkins Hospital Reports, 1900.



Sudamina occur frequently. Furunculosis was very common among my cases and was observed twelve times. Onychia occurred in 1 case. Erythema may occur early in the disease and sometimes confuse the diagnosis with scarlet fever, particularly if a pharyngitis be present. The erythema may be diffuse over the entire body, or it may be localized to some particular part, as, for instance, to the face; usually it is transitory. Urticaria may occur. In one of my own cases I noted it early in the second week. Bed-sores, multiple gangrene, and noma have been recorded. Henoch observed erysipelas of the face with the formation of bullæ in a boy of eight years.

A fine, branny desquamation occurs commonly during convalescence; it appears as an exfoliation of fine scales. It is not like the extensive exfoliation which occurs in scarlet fever, but resembles more that of measles.

*Lymph Nodes.*—Edsall has recently observed, in a considerable number of cases, that widespread though slight nodular enlargement occurs in typhoid fever, in some cases the enlargement is as marked as that which occurs in certain other infectious diseases and is usually looked upon as distinctive of those diseases. As a rule, the nodes are about the size of buckshot, but may be larger, and most easily palpated in the axilla and groin. The most active enlargement of the lymph nodes takes place toward the end of the disease or during convalescence.

*The Nervous System.*—The onset is frequently characterized by the occurrence of headache. In the severe cases delirium is often noted. The onset with meningeal symptoms is not infrequent. Some of the children are particularly apathetic after the onset of the disease; they sleep a great deal and refuse to answer questions. Younger children, those under the fourth year, may be very restless and cry almost incessantly. On the other hand, some of the severe cases in infants are characterized by a persistent stupor. Tremor of the extremities and the tongue is present in the severe types of the disease. Meningitis, brain abscess, and neuritis have been also observed. Neuritis affecting both upper and lower extremities, with sensory disturbances and atrophy, has been reported. The neuritis is often located in a plexus of nerves or in the root or trunk of one nerve only. The affection usually sets in with excessive pain, followed by numbness and partial paralysis. Sometimes complete paralysis of the muscles supplied by the affected nerve or nerves occurs.

Convulsions are usually of grave significance. Barthez reported 5 cases, of which 4 were fatal. Osler reports convulsions in a girl aged eleven years. After the temperature had been normal for eight days she had a convulsion on the left side which lasted for three or four hours. Recovery and consciousness were restored and she seemed quite herself, though somewhat confused. Eleven days later a tonic convulsion occurred. This also involved the left side and lasted five hours. After this seizure she was temporarily blind, but soon recovered. The whole condition gradually improved and in two months she was perfectly well.

Hemiplegia is very rare in children, in whom the condition is no



an infrequent accident of the specific fevers. Osler found no instance of hemiplegia as a result of typhoid in 120 cases of cerebral palsies in children. Wallenburg studied a series of 160 cases of hemiplegia and found that 4 occurred during the course of typhoid fever. The hemiplegia may occur during the second, third, or fourth week, or during convulsions. Aphasia usually accompanies the hemiplegia if it is on the right side. The cause of the hemiplegia is softening due to the thrombosis of the middle cerebral artery. Hemiplegia may also be due to abscess or embolism.

*Mental Affections Complicating Typhoid.*—Various mental disorders may follow typhoid fever, especially when the disease has been severe and protracted. A condition of imbecility and stupidity may last for many months. Melancholia is perhaps the commonest form of mental derangement. Maniacal attacks have been noted by Henoch in children four and seven years of age. Adams reports melancholia in two children. Both recovered after several weeks. He also reports two cases of mania, one in a child of seven and the other twelve years of age. One of my patients, aged nine years, became demented at the close of his febrile period. During defervescence and convalescence the knee-jerks and the abdominal and cremasteric reflexes are frequently increased, and ankle clonus is frequently elicited during the same period. These manifestations are probably more frequent in those of hereditary neurotic temperament. Post-typhoid insanity is now regarded as due to a nutritional disturbance; the result of nervous exhaustion and possibly insufficient food during the course of the disease. Nearly all of the cerebral affections following typhoid in children tend to recover, excepting those cases where hemiplegia occurs.

*Special Sense Organs.*—Of the organs of special sense, the ear is most often affected. Furunculosis of the external ear is observed; it usually occurs during convalescence and may be a part of general furunculosis. Otitis media is relatively frequent. It is not definitely determined how often the typhoid bacillus is the cause of these middle-ear affections. In the majority of cases the infection spreads from the nasopharynx through the Eustachian tube into the middle ear. Otitis media occurred five times in the cases which I observed. In one it was bilateral and four times it occurred only in one ear. As a rule, the middle-ear affections of typhoid are of a mild variety; sinus thrombosis, periostitis, and caries of the mastoid are rare complications.

Conjunctivitis sometimes occurs and occasionally its manifestations are of the severer kind. Curschmann suggests that the conjunctivitis may be due to the diminished activity of the lids and to the diminished secretion of tears. Late in the course of the disease, particularly during convalescence, foci of corneal inflammation may occur. It is very seldom that they lead to permanent disturbances of vision. Feebleness of accommodation as part of general postfebrile debility is a frequent symptom.

*Aphasia.*—Young children who have already learned to talk, frequently lose this power during an attack of typhoid fever, as in other

acute affections. Sometimes this aphasia becomes manifest during the first few days of the illness. During convalescence it is particularly noticeable. After the fever has disappeared, some children seem to have forgotten how to talk. Gradually, however, the ability to speak returns. Sometimes the aphasia is due to organic brain disease, as has been mentioned.

*Parotitis* is less frequently observed now, since cleansing of the mouth receives more attention. One gland is usually affected and later the other becomes involved. Parotitis occurs at the height of the typhoid at the end of the third week, but it may occur later, even during convalescence, and causes great pain. Parotitis has always been looked upon as a symptom of ill omen in typhoid fever. Suppuration sometimes takes place and may lead to thrombus of the jugular vein and the venous sinuses, or cause acute edema of the brain. Henoch has observed four cases of parotitis complicating typhoid.

*Bone and Joint Affections.*—In the inoculation experiments carried on by Chantemesse it was found that the typhoid bacillus could be traced to the medulla of bone. Abscesses are found most often in the tibia and femur; more rarely in the sternum, ribs, and other bones. Streptococci or staphylococci are most commonly found with a few typhoid bacilli. In children and young persons after convalescence one occasionally notices an exaggerated growth of the bones. Sometimes a circumscribed periostitis is recorded, which comes on without very great pain, and may undergo absorption. Keen distinguishes three forms of arthritis:

1. Typhoid arthritis proper. (a) Polyarticular variety. (b) The monarticular variety affects the larger joints, such as the elbow and shoulder, the ankle and knee, but more frequently the hip. As a rule, pain is slight, though it may be severe and prolonged. Swelling is observed in all joints except the hip and shoulder, where it is obscured by the muscular masses about these joints. Pus rarely forms.

2. Septic typhoid arthritis occurs rarely, is usually polyarticular, and is the result of a mixed infection with the typhoid and the pyogenic bacteria. It runs the usual course of similar septic inflammation and frequently terminates fatally in spite of all treatment.

3. Rheumatic typhoid arthritis is rare, it occurs where there was a previous rheumatic history, it is usually polyarticular and may be followed by a multiple ankylosis.

*The Hemorrhagic Form of Typhoid.*—Hemorrhagic eruptions may occur in the course of typhoid fever. As a rule, they appear in the neighborhood of the joints, and the exudation may be small in quantity, or quite large. Rarely does the tendency to bleed become general and result in hemorrhagic typhoid. I have seen a fatal hemorrhagic case in a little boy aged nine years. This variety is characterized by bleeding from various mucous membranes, usually in connection with a hemorrhagic skin eruption. This is a serious complication and nearly always fatal. One or more of the mucous membranes may be involved: there may be oozing from the gums, or epistaxis; hematuria and hemorrhage

from the vulva may be associated in the same individual. Autopsy reveals extensive internal hemorrhages, such as meningeal, pleural, peritoneal, intestinal, pulmonary—in fact, no tissue is exempt from this universal tendency to bleed. The patients are much prostrated; the tongue is usually heavily coated.

**Hemorrhagic complication** does not occur at any definite period of the disease. It is rare during the first week; it is most frequently observed when the fever is beginning to decline; it may occur during a relapse.

**Occurrence of Typhoid Fever with the Exanthemata.**—Typhoid fever may be associated with other acute infectious diseases. The presence of one infectious disease does not exempt the patient from another. If a typhoid-fever patient is exposed to another contagious disease he is not immune to it, but it may be considered that on account of lowered resistance his susceptibility to other infection is increased. Hence, typhoid may be associated with scarlet fever, diphtheria, measles, smallpox, chickenpox, whooping-cough, and sometimes malarial fever. These double infections occurred more frequently formerly, when febrile patients of all kinds were huddled together in large hospital wards.

**Duration.**—The duration of the disease depends upon its severity. Mild, uncomplicated cases may run their course in ten days. The severe cases in children as well as in adults may be protracted for many weeks or months. In cases in which relapse occurs, the disease necessarily runs a protracted course. Hensch stated that out of 80 cases, 11 lasted from seven to ten days; 26 from ten to fifteen days; 16 from fifteen to twenty days; 21 from twenty to thirty days; 6 from thirty to forty-nine days.

**Diagnosis.**—It is only recently, since the new laboratory methods of diagnosis are employed, that the recognition of typhoid fever is possible in nearly every case. In infants and young children the disease may closely resemble the ordinary intestinal infections so that the differentiation clinically is difficult. In both diseases intestinal disturbances, meteorismus, vomiting, and diarrhea may occur. Meningeal symptoms frequently mark the onset of typhoid fever, particularly in young children, and for the first few days of the disease it is very difficult, by our clinical methods, to differentiate meningitis from typhoid. Typhoid fever may be preceded by pneumonia; in these cases the recognition of the typhoid requires careful observation. In the mild typhoid of infants and young children the diagnosis from the clinical symptoms alone is difficult or impossible. In well-marked cases it is not difficult to diagnose the disease at the end of a week.

The splenic enlargement is an important sign. The spleen can usually be felt on the fourth to the sixth day of the disease. It was enlarged in 81 out of 90 cases which came under my observation. The splenic enlargement may lead to confusion in the diagnosis, as the spleen is enlarged in many diseases of childhood, particularly those which may be confused with typhoid fever, such as some of the acute and chronic



intestinal disorders of childhood—in acute miliary tuberculosis, sepsis and in other acute infections.

The *rose spots* are a valuable aid to diagnosis. They appear seldom in other diseases. They are present in rare cases of miliary tuberculosis and in cerebrospinal meningitis. They occur about the end of the first week. Their appearance in crops, their characteristic distribution over the body, and the fact that they are so common present make them important elements in the diagnosis. It is variously stated that roseolæ do not occur commonly in infancy and childhood; my own experience is that they occur quite as frequently as in adults.

*Laboratory Method of Diagnosis.*—The Gruber-Widal reaction for the diagnosis of typhoid fever is based upon the fact that the presence of the typhoid bacillus in the body produces substances which cause the agglutination of typhoid bacilli when allowed to act upon them. These agglutinins circulate in the blood and may be found both in the serum and in the corpuscles. Usually the test is made under the microscope. In case dried blood is used for the test, water should be added to the blood, so that it is half the desired dilution. (One drop of the fluid is mixed with an equal quantity of a culture of the typhoid bacillus in bouillon and placed in a hollow ground slide under the microscope. If the blood serum is used the typhoid culture and serum are mixed in the desired proportions and studied in the same way.

If the reaction is positive the typhoid bacilli are agglutinated in a short length of time which depends upon the dilution used and on the specific property of the serum. The following changes are noted under the microscope: The bacilli slowly lose their activity; they move about more sluggishly and finally collect into larger or smaller clumps. When the reaction is complete no actively motile bacilli are to be seen. At a dilution of 1 of blood serum to 40 of the culture a positive reaction should occur in about thirty minutes; 1 to 50 in forty-five minutes; 1 to 60 in one hour. The test may be performed without the use of the microscope by mixing typhoid serum of a patient with a typhoid culture in the proportions given and watching the reaction in a test tube. If the reaction is positive, the bouillon becomes clear and small whitish masses, due to the precipitation of the bacilli, are seen on the sides and bottom of the tube.

For all of the above tests a culture of the typhoid bacillus in bouillon should be employed. The culture should be not more than twenty-four hours old and grown in an incubator at 37° to 39° C. (98° to 102° F.). It should have been taken from a growth in agar. If the cultures are passed from one bouillon tube to a next for generation, autoagglutination occurs, and the bacilli cannot be used for the Gruber-Widal reaction.

Attempts have been lately made to use homogeneous emulsions of dead bacilli for the test. Unfortunately, the dead cultures cannot be used for more than a month or six weeks, because they are too rapidly agglutinated by normal blood serum.<sup>1</sup>

<sup>1</sup> Recently a "typhoid agglutometer" has been introduced by a well-known firm for the purpose of rapid diagnosis.



The Gruber-Widal reaction is present in less than 3 per cent. in persons not suffering from typhoid fever. It is present at some stage of the disease in 95 per cent. of typhoid patients. It is most often first obtained at the end of the first or the beginning of the second week. It may be delayed, however, until the sixth or seventh week, or until all symptoms of the disease have disappeared.

In persons in whom icterus is present from any cause, a positive Widal reaction occurs, according to Köhler. Icteric blood possesses strong agglutinating power toward typhoid bacilli, and hence the Widal reaction would be of no value in persons suffering from jaundice. On account of the persistence of the Widal reaction for long periods of time after typhoid, the test may be positive in a person who has passed through, but is not suffering from, an attack of typhoid fever.

On account of the lateness of the appearance of the Gruber-Widal reaction in many cases of typhoid fever, another test has been advocated—i. e., the use of blood cultures made from freshly drawn blood. With improving technique in bacteriology from year to year, typhoid bacilli have been found in greater numbers in the circulating blood of typhoid patients. Busquer found them in every one of 43 cases examined by him—in 22 during the first week of the disease, and very often before the agglutination reaction was present. The technique is very simple. The blood is withdrawn by puncturing a prominent vein in the forearm, and from  $\frac{1}{2}$  to 2 c.c. of this blood is introduced into 50 to 150 c.c. of bouillon. In twenty-four hours the bouillon is seen to be turbid, and if typhoid bacilli are present they may be agglutinated by a known typhoid serum.

The urine is estimated to contain typhoid bacilli in 20 per cent. to 50 per cent. of the cases. The feces always contain them early and in large numbers. However, the separation of the bacilli is difficult on account of the great number and variety of micro-organisms. The method of Chantemesse, known as the gelodiagnosis, depends upon the fact that only the colon and typhoid bacilli resist the action of dilute carboic acid. The details of this method require laboratory technique. The feces are inoculated into bouillon. To this bouillon a few drops of typhoid serum are added, which cause the precipitation of typhoid bacilli if present. The precipitate is then plated on alkaline gelatin to which 3 per cent. carboic acid has been added, and which has been colored slightly by litmus. The colonies of typhoid bacilli are distinguished from those of colon bacilli by remaining bluish, while the latter, by forming lactic acid, color the litmus red. The final test for the presence of typhoid bacilli is their agglutination by a known typhoid serum.

**Differential Diagnosis. Paratyphoid Fever.**—Since the introduction of the serum reaction a certain number of cases have come to light which resemble typhoid, although the Widal test remains negative throughout the entire course of the disease. Closer examination of the blood, feces, and urine yields a bacillus resembling that of typhoid, but not identical with it. Clinically the cases closely resemble true

typhoid fever. As a rule, they are mild and the prognosis is favorable. The diagnosis is made by finding the paratyphoid bacillus in the blood or by the agglutination of paratyphoid cultures with the blood of the patient. Coleman and Buxton report the case of a child, aged seven months, from whom the paratyphoid bacillus was isolated. The reported cases are still few in number, so that a larger experience and more definite knowledge is needed on the subject.

*Tuberculosis.*—The differentiation between typhoid fever and acute miliary tuberculosis, particularly in children, may give rise to great difficulty, and is mentioned in the article on Tuberculosis. The prodromata of typhoid fever are very short; in miliary tuberculosis they are very long and are marked by occurrence of emaciation. In both diseases splenic enlargement occurs; high fever is common to both; in rare instances an eruption occurs in miliary tuberculosis, which may be absent in typhoid fever. In both diseases bronchitis and meningeal symptoms may occur. The history should be carefully inquired into. In some of the tuberculous cases a history of infected lymph nodes or joints and a previous or existing affection of some part of the lungs may aid in establishing the tuberculous nature of the disorder. A history of measles or whooping-cough, in which the pulmonary affection has not cleared up, or the continuance of a persistent cough, speaks somewhat in favor of the tuberculous nature of the disease. The fever is not of diagnostic value. In miliary tuberculosis the fever may be remittent in character. The physical examination of the lungs is, as a rule, negative during the first stages of miliary tuberculosis; at the most, there is evidence of a slight bronchitis. Notwithstanding the slight physical findings, dyspnea and cyanosis occur very early and the patients suffer from an annoying dry cough. Rarely during the first stages of miliary tuberculosis tubercles appear on the surface of the pleura or pericardium and give rise to friction rubs. In cases where this occurs, tuberculosis may be suspected. In young children the condition of the pulse is not as important as in older children. It has already been pointed out that the pulse rate is rapid in typhoid of children, though not as marked as in tuberculosis. Tuberculous meningitis, as well as the meningeal symptoms of typhoid fever tends to retard the pulse during the early stages. For this reason the pulse as a differential sign is of relatively little value in young children. Ophthalmoscopic examination should be made in doubtful cases. If tubercles appear upon the choroid the diagnosis of miliary tuberculosis may be made with certainty. They are sometimes observed very early or they may appear a few days before the fatal termination; one or both eyes may be affected and no disturbance of vision be produced by the tubercles.

The sputum may be examined for tubercle bacilli; but it is to be remembered that the sputum may be difficult to obtain, and the sputum of patients with miliary tuberculosis very seldom contains tubercle bacilli. The diazo reaction is found in both acute miliary tuberculosis and in typhoid fever. Fortunately, the Widal reaction, the examination of the

blood, urine, and feces for typhoid bacilli make the diagnosis possible in the most difficult cases.

Tuberculous meningitis may run a febrile course lasting for ten days or two weeks, which may simulate a typhoid. In tuberculous meningitis the headache is usually violent. The patients soon fall into a comatose condition, the pulse becomes slow and irregular, the abdomen retracted; while in typhoid it is usually distended and tympanitic. Sometimes the meningeal symptoms do not appear for one or two weeks. These are cases of acute general miliary tuberculosis which terminate in meningitis. It has already been noted that meningeal symptoms or meningismus, so-called, may occur at the onset of typhoid fever. This condition usually disappears at the end of the first or at the beginning of the second week. Cases of true typhoidal meningitis have been reported.

It is sometimes difficult, especially in infancy, to differentiate intestinal infections with a constant fever from typhoid fever. In cases which run a protracted course and which are marked by fever, diarrhea, and tympany, repeated Widal examinations should be made, or the urine and feces examined. The leukocytes should be counted; a leukopenia would indicate typhoid.

Influenza may be confused with typhoid fever. In young patients influenza sometimes runs a course which is characterized by high fever and exhaustion, without other definite symptoms. The fever in these cases may be remittent or intermittent. The pulse and respirations are rapid in influenza. The existence of an epidemic of influenza, the general course of the disease, and the serum test for typhoid are of great assistance in differentiating the two disorders. Influenza bacilli are sometimes found in sputum and on mucous membranes.

Pyemia may be mistaken for typhoid fever, especially in cases where the original focus of infection is deep-seated. Fagge observed two cases of pyemia in which there was latent abscess of the lumbar or dorsal vertebrae. In pyemia the temperature is more irregular than in typhoid, and profuse perspiration and chills are important distinguishing elements. Leukocytosis is marked in pyemia.

In young individuals who present obscure and complex typhoid symptoms the epiphyses of the bones should be carefully examined for localized edema, redness, and pain due to osteomyelitis. In osteomyelitis there is usually a decided leukocytosis; in typhoid, a leukopenia.

Malaria may be mistaken for typhoid fever. The reverse is also true; typhoid fever may be mistaken for malaria. This difficulty arises only in those regions where malarial fever is prevalent. The blood examination for plasmodium and the laboratory tests for typhoid fever are the most valuable and certain methods of differentiation. Quinine may be administered as a therapeutic test.

Epidemic cerebrospinal meningitis or acute purulent meningitis must sometimes be differentiated from typhoid fever. Laboratory tests are the most important in making the differentiation. The Widal test and the leukopenia of typhoid, the leukocytosis of epidemic meningitis, and



the discovery of diplococci in the cerebrospinal fluid are points which determine the diagnosis.

*Appendicitis* and typhoid fever are sometimes mistaken for each other. Without pain at some time in the course of the disease, there can be no acute surgical lesion of the abdomen (Richardson). The diagnosis of appendicitis is made from the local symptoms—pain, rigidity, temperature. The onset in appendicitis is abrupt. In place of gurgling in this region there is a sense of resistance on palpation, and sometimes dullness on percussion. In typhoid fever there is more or less temperature with pain, but without rigidity or tenderness.

**Prognosis.**—This varies with the epidemic, with the severity of the disease, and the previous health and resistance of the child. Prognosis is grave in infants. Poorly nourished children, or those who have been debilitated by constitutional or acute diseases, have a less favorable prognosis than those who are robust. The mortality statistics vary within wide limits. Henoeh in 375 cases had a mortality of 14 per cent.; Blackader in 100 cases lost only 1; J. P. C. Griffith reports a mortality of 3 per cent.; Koplik reports a mortality of 8.7 per cent.

In my own experience, in the first series of 90 cases, 2 died—2.2 per cent.; 1 died of bronchopneumonia and exhaustion; another, twenty-one months old, died as a result of multiple gangrene. Of the last 110 cases, 4 died, representing a mortality of 3.66 per cent. Of these, 2 died of intestinal perforation, 1 of bronchopneumonia, the third from repeated intestinal hemorrhage, and the fourth from severe general hemorrhage.

**Treatment. Prophylaxis.**—The contamination of drinking-water being the most prolific source of typhoid infection, the disease can be almost eliminated from cities by careful regulation of their water supply. Similarly, the delivery of milk by dealers in whose families typhoid fever exists should be absolutely prohibited. If a nursing mother is taken ill with typhoid the infant should be weaned. The antityphoid vaccination of Wright has been employed in the English army, and it is claimed that the occurrence of the disease and the mortality have both been greatly reduced.

Great care must be used, in every household in which typhoid fever exists, that the discharges of the patient do not infect healthy individuals. Feces and urine, especially, must be thoroughly disinfected. The best antiseptics for this purpose are crude carbolic acid, 1:10 solution, or chlorinated lime. A pint of the disinfectant should be in the bed-pail before use. All instruments and utensils coming in contact with the patient should be similarly disinfected and cleansed, and in hospital should not be used for other patients. The bed-linen of the patient must be disinfected before being washed. These measures should be continued for at least ten days after the temperature has been normal.

In communities in which typhoid fever is endemic or epidemic, the drinking-water should be boiled. Persons changing their residence from a locality in which the disease has not existed to one in which the disease is common appear to be especially susceptible to infection.



*General Management.*—Good nursing and careful hygienic management are the most important elements in the treatment of typhoid fever. Older children should be placed in bed and kept there constantly. The use of the bed-pan and the urinal must be insisted upon. In infants and younger children this method is not practicable. The restlessness in bed is so great that it is necessary at times to lift them up so that they may be held in the nurse's or mother's arms. The nurse should note minutely the various symptoms which occur in the progress of the case, particularly those which arise in the absence of the medical attendant. The nursing record should be carefully kept; the temperature should be taken by rectum at intervals of three to four hours; the pulse and respirations should be taken preferably when the child is asleep. The sick-room should be large and well ventilated; the temperature should not be too warm, not more than 65° to 70° F. during the day, and a lower range at night. Screens placed about the bed protect the patient from draughts and direct sunlight, and in this way add to his comfort. A single bed away from the wall may be approached from either side. A woven-wire spring mattress, over which is placed one of hair and upon this a double blanket, constitutes the best bed for a prolonged illness. A rubber cloth should be spread under the sheet and the sheet should be kept smooth to prevent the formation of bed-sores.

The position of the patient in bed should be changed from time to time. A change of position tends to prevent hypostatic congestion of the lungs.

The mouth and tongue of the little patients should be kept scrupulously clean by the use of a mild antiseptic wash, such as a solution of boric acid. The teeth also should be cleansed. These precautions with reference to the mouth prevent stomatitis and possibly other infections.

*Diet.*—In children under two years of age the diet should be managed somewhat after the plan that is employed in the gastroenteric infections of infancy. If the stools are thin, frequent, and contain mucus, or are fetid, the use of milk should be discontinued for a time and one of the cereal waters like barley, arrow-root or rice-water should be used. After the stools show a tendency to become more nearly normal, milk may be resumed, though it is better to give it diluted, peptonized, or at less frequent intervals than is usual during health. The quantity of milk may also be reduced, and it may be diluted with plain water or with one of the cereal waters. From five to six ounces may be given every three or four hours.

If the stomach is irritable and food is retained with difficulty, or if the patient has no desire for food, small quantities of nourishment may be given at short intervals. In cases where the patient has been restless he should not be awakened from a refreshing sleep because the time for feeding has arrived. During the night the food should be given less often than during the day. When digestion is weak the food should be peptonized. Egg-albumen water is often useful. The patient should be given freely of plain water.

In older children the dietetic management does not differ essentially from that of adults. The diet should be fluid and easy of digestion.

Milk which is clean and fresh should be the principal article of food. Dilution or peptonization makes it more easy of digestion. Beef-juice, animal broths, cereal waters, and strained gruels may also be required for some of these children.

I have still to be convinced that the general use of eggs and other nitrogenous or carbohydrate foodstuffs is desirable during the height of typhoid fever. Whether it has been mere coincidence or directly the result of the food, nevertheless, rise in temperature, a feeling of abdominal discomfort, and general aggravation of the symptoms have followed most of my attempts at complex feeding.

*Alcohol* should not be administered in a routine manner. It should be held in reserve as a remedy of value to combat the effects of the typhoid toxemia. In cases of great prostration with heart weakness, whiskey or brandy may be given in 20 or 30 drop doses every two or three hours, to children under two years of age. In older children the dose may be proportionately larger.

*Diet during Convalescence.*—When the fever has fallen to normal the patient recovers from the apathetic state which is common to nearly all cases of typhoid fever. He usually announces the return of his appetite or else complains of hunger. It is a safe rule to postpone the resumption of the usual dietary régime until the seventh or eighth day of normal temperature has elapsed. During these seven or eight days the older children may continue the use of the broths, to which some cereal like rice or barley may be added; orange-juice may be given during this time and the strained gruel which was permitted during the course of the fever may be thickened somewhat. Beginning the second week of convalescence, soft-boiled egg, milk-toast, and baked custards may be allowed. Later on, scraped beef slightly broiled; farinaceous foods, like rice, tapioca, and farina; tender sweetbread or fish, and potato which has been baked and mashed may be gradually added to the diet.

*Hydrotherapy.*—It is a pretty general experience that children bear the cold bath badly; that is, water which has been reduced to 70° or 75° F. In recent years the children that have come under my care have been given warm baths for a pyrexia of 103° F. They are placed in the tub with water at a temperature of 88° to 90° F. A hammock is suspended over the tub and when a child is placed in the bath it reclines comfortably on the hammock. Tubbing is continued for five or ten minutes, in water between 85° and 90° F.; the patient's temperature is usually reduced 2° and the pulse and respirations fall accordingly. The children are constantly rubbed while they are in the water. This is a detail often overlooked. In cases where the temperature decline was not satisfactory, or in those cases where the fever was unusually high, it was found that if the children were left in the bath ten or twelve minutes a greater reduction of temperature could be obtained. We also observed that the bath-water was raised 2° after the completion of the bath. Water that was 90° F. when the child was placed in it was raised to 92° F. when the bath was completed.

In mild cases sponging with tepid water may be employed and a thin film should be left on the body surface. It is considered that the evaporation rather than the temperature of the water is effectual in cooling the body. The use of guaiacol externally, for reducing temperature, is not applicable as an antipyretic measure among infants and young children.

*Important and Symptomatic Treatment.*—The mortality rate of typhoid fever has steadily fallen, although the use of medicines in a routine way has steadily decreased. With an experience of 200 cases, carefully observed and tabulated, I rarely found it necessary to administer drugs save as they were indicated for some special symptom. Antipyretic drugs of the coal-tar series have no favorable influence on the course of the disease, and their use may be very well dispensed with.

The *antiseptics*, of which a large number have been employed, have not won a permanent place in typhoid fever therapy. This group of drugs, whether it be calomel, salol, iodine, carbolic acid, or any of the host of remedies which have from time to time been advised, have no influence on the duration, course, or mortality of the disease. Typhoid fever is not a local disease of the intestinal tract, but is a systemic disorder, with bacilli and toxins of the disease circulating freely in the blood and carried to the most remote tissues and organs of the body. Antiseptics, to be effectual, must be general, not local; hence the inefficiency of intestinal antiseptics.

The various methods of *serum* and *culture treatment* are as yet of no practical value in the treatment of the disease. E. Fränkel described a method of treatment by deep subcutaneous injections of sterilized cultures of typhoid bacilli grown on thymus bouillon. He thought that the treatment was effective, although neither complications nor relapses were prevented. Rumpf used cultures of the bacillus pyogenus prepared in a similar way in two patients, both of whom died—one of pneumonia, the other of intestinal hemorrhage.

Chantemesse's serum was employed in fifty children. No local or general disturbances resulted from the injection. No striking cures were effected with this serum.

At present no specific typhoid fever serum is available.

*Treatment of Special Symptoms and Complications.*—Headache, as a rule, requires little special treatment. The nurse should be directed to keep the patient quiet; an ice-bag may be applied to the head. The headache generally disappears spontaneously at the end of the second week. With the treatment by baths they are rarely severe. Bromide of sodium may be used in persistent cases.

For the restlessness of infants as well as of older children, relief is nearly always obtained by bathing and the use of the ice-bag. Bromide of sodium, 0.13 to 0.3 gm. (2 to 5 gr.), small doses of trional, 0.06 to 0.18 gm. (1 to 3 gr.), may be used if a sedative is needed.

*Vomiting.*—This does not occur frequently during the height of the disease. It usually indicates that the food is disagreeing, and the best plan is to stop all food temporarily. Small pieces of ice may be admin-



istered, or at other times small doses of bismuth may be given internally. Teaspoonful doses of carbonated water or very hot water are sometimes useful.

Most of the patients show a tendency to *constipation*. A daily enema of normal salt solution is indicated in obstinate cases; mild soapsuds or oil injections may be used. The use of the long rectal tube, as a rule, is unnecessary. The small rubber point of the fountain syringe is sufficient; a rounded glass point is preferable, as it can be disinfected and boiled. These patients should have one bowel movement a day. If fecal impaction occurs, feces must be broken up and extracted by the fingers after injections of sweet oil or ox-gall. Laxatives are rarely necessary; if used at all, only the milder ones, such as milk of magnesia, 3.75 to 7.5 c.c. (1 to 2 drams), or castor oil, should be used. If three or four stools occur during the twenty-four hours, little or no medicinal treatment is called for. If this number is exceeded, some measure should be employed to control the diarrhea. Subcarbonate or subnitrate of bismuth may be given in 0.6 gm. (10 gr.) doses to children of two years or over. The stools should be inspected carefully; if they contain undigested food masses, the milk should be diluted or discontinued temporarily. Tanningen or tannalbin in 0.06 to 0.18 gm. (1 to 3 gr.) doses may also be given to children under three years of age. In obstinate cases small doses of opium may be required. The medicinal treatment of the diarrhea should not be carried too far; obstinate constipation sometimes results, which may cause pronounced toxemia and tympany.

The dietetic treatment of *tympanites* is similar to the treatment of diarrhea. Warm compresses or turpentine stupes made by mixing one part of spirits of turpentine with six parts of sweet oil may be applied. The abdomen is covered with a thick piece of flannel which has been dipped in hot water, wrung out, and when sufficiently cool applied to the abdomen and covered with oiled silk. The rectal tube may be used with caution; a rectal injection often brings relief. The meteorism is not infrequently the result of a fermentation process in the bowels. Salol, the carbonate of guaiacol, or charcoal may be tried in these cases. If severe abdominal pain occurs, the use of an opiate, preferably paregoric, may be necessary.

Upon the first occurrence of a *hemorrhage* from the bowels the patient should be enjoined the utmost quiet. The baths should be discontinued and the food limited to the smallest possible amount. During the first few days a few teaspoonfuls of cold milk should be given and the patient may be permitted to swallow small pieces of ice. An ice-bag should be applied to the abdomen. Opium in the form of paregoric or Dover's powder should be administered in sufficient quantity to quiet peristalsis. In giving any of the opium preparations, their effect should be watched, and sufficient time should be allowed to elapse to judge of the action of the initial dose before a second dose is given; 0.001 to 0.0016 gm. ( $\frac{1}{80}$  to  $\frac{1}{40}$  gr.) of morphine, combined with 0.00021 gm. ( $\frac{1}{800}$  gr.) of atropine may be given to children three years



**1041.** A solution of 2 per cent. gelatin may be administered by mouth or by rectum to control hemorrhage. The use of gelatin hypodermically is unsafe, as it may cause toxemia, and it has caused tetanus in a number of cases. Severe hemorrhage is sometimes followed by anemia or collapse. In such cases hot saline enemata or the infusion of normal salt solution under the skin or into the veins is indicated.

*Heart weakness* should be treated by stimulants. It is sometimes due to change in the myocardium or to general prostration. These cases are treated by the use of brandy or whiskey, digitalis, strophanthus, or strychnine, either by mouth or hypodermically; 0.00013 gm. ( $\frac{1}{500}$  gr.) of strychnine may be given three or four times daily to a child one year of age; 0.016 c.c. to 0.12 c.c. ( $\frac{1}{4}$  to 2 min.) of tincture of strophanthus or digitalis is the usual dose for a one-year-old child. When collapse occurs, nitroglycerin is indicated. For a child one year old the dose is 0.00026 to 0.00013 gm. ( $\frac{1}{2500}$  to  $\frac{1}{500}$  gr.).

*Parotitis* should be treated by the application of an ice-bag to the swollen gland. If fluctuation occurs it should be incised. *Furuncles* should be treated by incision.

Pulmonary complications, as bronchitis and pneumonia, and also neuritis and joint affections should be treated on the lines which are indicated in these disorders.

If the attending physician suspects that perforation is threatening, or if it has actually occurred, the case is distinctly a surgical one and operative procedure should be instituted if the diagnosis is reasonably certain and the condition of the patient permits.

## MALARIA.

By JOHN RUHRÄH, M.D.

Malaria is an infectious disease caused by the hemacytozoön described by Laveran. It is characterized by paroxysms of intermittent fever of a quotidian, tertian, or quartan type. In other cases there is a remittent fever. There may be rapidly fatal or pernicious forms or there may be a cachectic condition with anemia and an enlarged spleen.

**Etiology.**—Owing to a better understanding of malaria and its causes, and to the drainage of marsh land, the disease is becoming less and less frequent in civilized countries. A transmitted immunity has been regarded as lessening its prevalence.

In this country the disease is found in a number of places. In many localities along the Atlantic coast and throughout the Southern states it is seen with comparative frequency, but in the Northwest and West cases are more unusual. In the tropics it is most frequent in spring and fall, while in summer and winter there is but little. In the temperate climates a few cases may be seen in spring, but the majority are observed in August, September, and October, and even in November.

Negres are supposed to be less susceptible than the white races.

It is undoubtedly true that the disease may be contracted *in utero*, when the mother has the disease in a severe form. This is usually, however, a very rare occurrence.

**Mode of Infection.**—The ordinary mode of infection is through the bite of a certain genus (*Anopheles*) of mosquito, which acts as the intermediate host for the malaria parasite. So far the parasite has been found only in these mosquitoes and in man.

**The Parasite.**—The parasite is a hemacytozoön, or a parasite which attacks the red blood cells. There are a number of different hemacytozoa found both in man and in animals.

The hemacytozoön of malaria was first discovered by Laveran in 1880; subsequently it was described very accurately by Celli and Marchiafava; then Golgi noted that the fever and the segmentation occurred at the same time. In this country the organism has been thoroughly studied by Osler, Thayer, and many others. Manson formulated the theory that the infection was due to the mosquito, and Ross demonstrated the development of the parasite in the body of the intermediate host (mosquito). The disease was produced in young Manson by letting infected mosquitoes bite him.

There are three forms of the parasite known—the tertian, quartan, and the estivoautumnal.

1. The *tertian* parasite completes its cycle of development in the human body in forty-eight hours. When first noticed it is a small, oval or irregular-shaped mass, without any pigment, in the centre of a red blood cell. This is about  $2\mu$  in diameter. (A red blood cell is about  $7\mu$  in diameter.) It looks very much like the spore forms seen during the chill, and the parasite looks as if it were covered by part of the red cell. This develops rapidly and in a few hours pigment may be seen. This is fine, granular, and brown in color. The pigment is arranged about the periphery of the parasite and there is a clear area, partly transparent and partly milky white which contains no pigment. There is distinct amoeboid movement, protrusions being put forth and then withdrawn. As development goes on the red blood cells containing the parasite seem larger than the others and the color is paler, as if the hemoglobin had been absorbed. The pigment in the parasite increases. Just before the chill the parasite fills the most of the red blood cell. The pigment becomes grouped in the centre of the parasite and the protoplasm splits from the centre to the periphery into from fifteen to twenty segments, the lines of fission being like spokes of a wheel. These segments are the so-called spore forms and they enter the red blood cells and go through another cycle of development. Some of the full-grown parasites do not segment, but remain with actively moving pigment granules. These are the so-called gametocytes or sexually differentiated parasites.

2. The *quartan* parasite is quite rare in the United States. It takes seventy-two hours to complete its cycle of development in its host and, hence, the chill and fever are seen on every fourth day. The early stages are like the tertian. The granules are larger and darker, however,

and there is not so much movement. By the third day the parasite is usually quite still and the pigment at the periphery. The red blood cell is of a dark yellowish-green or brassy color. On the fourth day the pigment moves toward the centre and is seen in radiating lines which give the parasite a rosette appearance. The parasite segments into from six to twelve spore forms. There are some which do not segment (gametocytes) as in the tertian form.

3. The *estivoautumnal* parasite is the parasite of the more irregular fevers. Its cycle probably takes twenty-four to forty-eight hours, and after a week there are seen curious crescentic forms. The parasite is smaller than the preceding, being about half the size of the red blood cell. There is but little pigment. Usually there are seen small hyaline bodies with one or two pigment granules near the periphery. The later stages of development must be studied in the internal circulation, as in the blood of the spleen. The corpuscles containing the parasite shrink and are of a distinct yellowish-green color. After about a week large, oval or crescentic forms are seen. These have pigment in the centre and often have the remains of a red blood cell adhering to them. They are the sexually differentiated forms or gametocytes.

The gametocytes of all the forms do not develop any further in the human blood, but they do upon the slide or in the intermediate host. The male parasite gives off flagellæ which enter the body of the female. If this occurs in the stomach of a mosquito the fecundated parasite enters the wall of the stomach and undergoes further development. Two days after the mosquito has bitten the person whose blood contains the malaria parasite, small refractive bodies may be seen in the wall of the stomach of the mosquito. In about a week these have developed, become striated, and burst into myriads of spindle-shaped sporozooids. These get into the poisonous salivary glands of the mosquito and, escaping by the ducts, infect the individual bitten. On entering the blood of the host these sporozooids develop into young parasites.

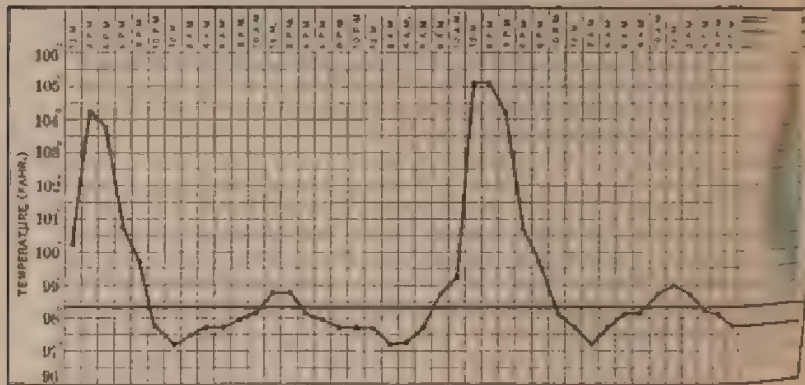
As stated, the genus of mosquito which acts as intermediate host is the *anopheles*, of which numerous species have been described. The common genus of mosquito is the *culex*. The two are easily distinguished. The *anopheles* has two large palpi, one on either side of the proboscis. The wings are mottled. When on the wall or ceiling the body is inclined away from the wall at an angle of forty-five degrees or more. The harmless *culex* has small palpi, no spots on the wings beyond the veins, and the body is parallel to the wall and usually the two posterior legs are crossed over the back. The *culex* is common in the city, while the *anopheles* is found in the country.

**Pathology.**—Malaria is rarely fatal in infants and children in this country. The fatal cases are the so-called pernicious forms. The lesions in malaria cachexia are sometimes seen when the child dies of some intercurrent affection. The changes are much the same as in adults. In the pernicious forms there is enlargement of the spleen and liver. The blood corpuscles are destroyed and the serum of the



blood may be tinged with hemoglobin. In the chronic cases there is pigmentation of the spleen and liver and of many other tissues, as in the brain and kidneys. Nephritis may be found occasionally.

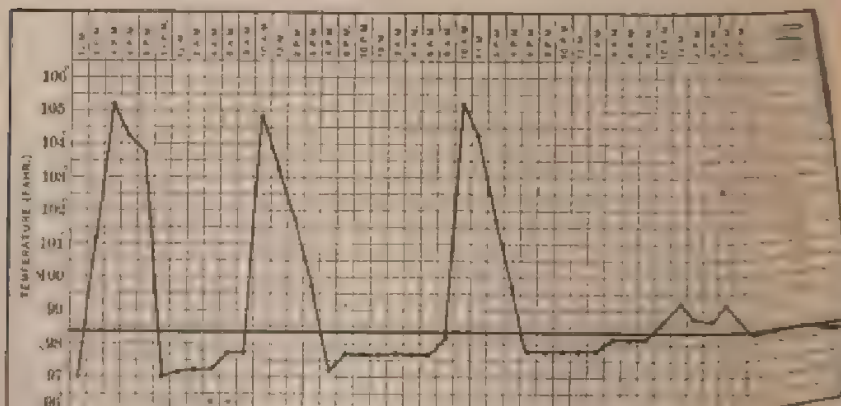
FIG. 98



Temperature chart of tertian type of malaria.

**Symptomatology.**—The younger the child the more apt is the disease to be irregular in its forms. After five or six years of age the adult type of the disease is met with. If the infection is with the tertian type of organism the paroxysm comes on every other day (Fig. 98). If, as most usually happens in young children, there is a double infection with the tertian organisms, and they mature on alternate days, the paroxysm will come every day, the so-called quotidian fever (Fig. 99). If the quartan

FIG. 99



Temperature chart of quotidian type of malaria.

organism is found the paroxysm will come every fourth day. In this country the quartan parasite is so rare as to be practically disregarded. If the infection is with the estivoautumnal parasite there may be



**intermittent fever** of a daily or of an irregular form, or there may be a **remittent fever**, the temperature going up and down, but seldom if **ever** reaching normal (Figs. 100 and 101). This may be mistaken for **typhoid** or may go under the name of bilious fever or typhomalarial fever or under some local designation. In the pernicious form of **malaria** the parasite found is of the estivoautumnal type.

**FIG. 100**

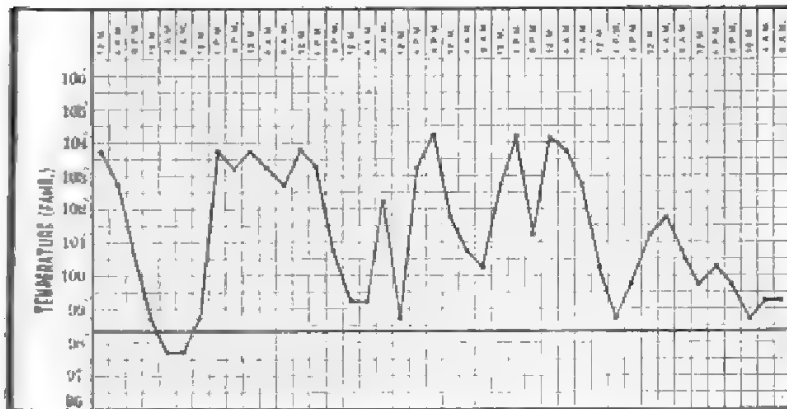
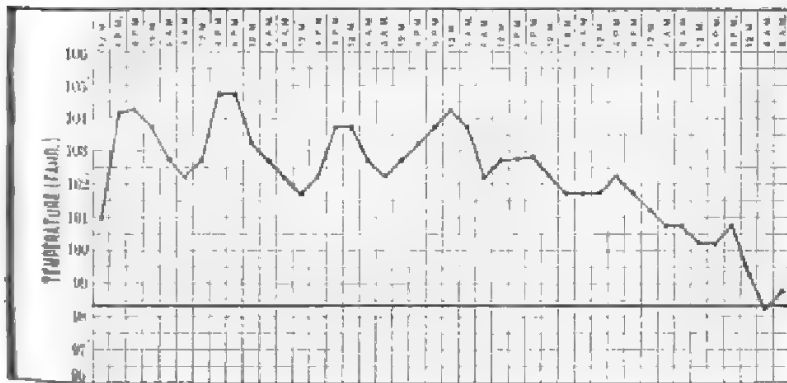


FIG 101



#### Temperature charts of estivoautumnal type of malaria.

**The Malarial Paroxysm.**—In older children the paroxysm resembles the adult type. The child may have been feeling quite well previously, or may have been complaining of not feeling as well as usual. The attack starts with a chill, which, in younger children, may be replaced with a convulsion. This may be preceded by stretching and yawning, and it is not uncommon to have an attack of vomiting at the outset or even several loose bowel movements. The child complains of feeling

cold and looks blue and peaked. There may be shaking even of a severe form. The child wishes to be covered up in bed. The hands and feet are cold, the nails and lips blue, and cyanosis may be very marked. The temperature, especially if taken by the rectum, is elevated. This cold stage lasts from a few minutes to an hour and then there is a stage of high fever, the feeling of cold giving way to one of heat, and the child, while still feeling badly, does not complain as much as before. There is usually intense thirst and headache. The temperature is about 104° or 105° F., but may even go above that. After from half an hour to four or five hours of high temperature the fever breaks. There may be a profuse sweat, but this is not as common in children as in adults. The temperature drops to normal or below. The child feels weak, but after a short time feels about as well as usual.

Under five years of age the attacks are not so typical. The chill may be entirely replaced by a convulsion, by an attack of yawning or stretching, or by an attack of vomiting or of diarrhea. There may be simply an attack of cyanosis without anything else of note. The lips and nails may be blue and the hands and feet cold. The expression may be noticeable, the face being pinched and the eyes sunken. The temperature if taken is found to be high. In some the chill may be replaced by a cold nose or some such trifling symptom, and in many there is nothing definite noted. In young infants the fever is quotidian and this is usually due to a double infection with a tertian parasite, although the estivoautumnal may be found. A single infection with a tertian parasite is not so common.

Of greater importance, as they are more apt to be misinterpreted, are the *irregular forms*. The child may have no paroxysm at all and the fever may be of a very irregular type, so that the diagnosis will have to be made upon other things. The fever may be intermittent in type, going up and down at irregular intervals, and it may be overlooked unless the temperature is being taken at short periods. I have seen children whose morning and evening chart did not show anything, but in whom fever of an irregular type was revealed by a two-hour or four-hour chart. Even a four-hour chart may not indicate it. Another form which is apt to be mistaken for typhoid is the remittent type. In this the child has a continuous fever, the temperature going up and down, but seldom if ever touching normal. There are also subacute forms in which the child is never very sick, but is more or less listless, well one day and sick the next, sallow and anemic, and, as in all the other forms, with an enlarged spleen.

If the disease is untreated it apparently gets well of its own accord after several weeks or more, to recur at some future time, or it leaves the child with the subacute form or with a condition known as a malarial cachexia. Repeated attacks of the disease will produce the cachexia.

*Malaria Cachexia.*—These cases may be mistaken for some form of anemia and the real cause be overlooked. There is a severe grade of secondary anemia. The child is pale, sallow, and the skin has a yellowish tint. The eyes are sunken and the facial expression is

begone. The tongue is coated and the appetite is lost. There is liable to be an indigestion with attacks of vomiting. Constipation is the rule, but there may be diarrhea. The spleen is large and hard. In some instances the spleen may be enormously enlarged; the liver may also be enlarged. There is slight edema about the feet and ankles. There is a tendency to hemorrhage. The urine may contain blood.

*Pernicious Malaria.*—This is rare in children in this country. The attack generally starts with vomiting, a convulsion, and high fever. In some instances the convulsions continue or the child becomes comatose. Cases have been reported where the coma came on with each paroxysm and disappeared when it was over. The diagnosis is by blood examination, and unless treated with subcutaneous or intravenous injections of quinine death occurs.

*Associated and Complicating Conditions.*—The enlargement of the spleen is one of the most noteworthy. I have never seen a case of malaria in a child where the spleen could not be palpated. The spleen seems to get large during the paroxysm and may be felt just below the margin of the ribs. Usually it is constantly enlarged. In the chronic forms the spleen may reach an enormous size and be mistaken for some other condition. Unless very chronic the spleen returns to normal or nearly normal size when the child is treated with quinine. In the very large spleens the size is greatly diminished, but treatment will not cause it to return to the normal size. It is surprising how rapidly the spleen may diminish in size under quinine.

Anemia is marked owing to the destruction of the red blood cells. There is a slight hematogenous jaundice when the disease is severe or where it has persisted any length of time. Leukocytosis is rare in malaria.

Herpes is frequently seen about the lips. Coryza is not uncommon and cases have been observed where a coryza may take the place of the sweat. Bronchitis is frequently seen in malaria. Pneumonia of malarial origin was formerly described, but probably is merely a complicating affection. What does occur in children is a severe congestion of the lungs which gives marked signs. This may be limited to one lobe and be mistaken for a beginning pneumonia. It clears up entirely in a day or two.

Albuminuria may be met with and occasionally nephritis. The stomach and intestines are usually more or less irritable. Vomiting is easily excited and diarrhea not infrequent. In chronic malaria there is apt to be constipation.

Among other symptoms that may be observed are headache and drowsiness. Neuralgia is frequently seen. Multiple neuritis of malarial origin has been described, as has also spasmodic torticollis.

*Diagnosis.*—This is best made by an examination of the blood. Both fresh and stained specimens should be studied. It requires considerable practice to become expert in the diagnosis of malaria from blood slides.

If malaria is suspected repeated examination should be made. I have seen the organism found after twenty or thirty trials. Quinine

should not be given if the blood is to be examined, for it tends to drive the parasite out of the peripheral circulation. In the severe cases the organism is usually found on the first examination. Quinine should, however, not be withheld for any length of time in any case if there is good reason to suspect malaria. A fever which responds promptly to quinine is probably malaria. A fever which does not respond promptly to quinine is something else.

From typhoid fever the Widal agglutination test is of value.

In general it may be said that the diagnosis of malaria may be suspected from what has been said of the paroxysm, the fever, the anemia, the enlarged spleen, and the cachexia.

If the spleen cannot be felt in a child some other explanation of the fever should be sought for.

**Treatment. Prophylactic.**—Much can be done in malarious districts in doing away with mosquitoes and in protecting children from their bites. Drainage of marsh lands and the use of petroleum on the breeding places are both efficient. Screens in the windows and doors of houses or mosquito nets over the child's crib should be used. If out-of-doors at night the face should be covered with a veil and the hands with gloves. Oil of pennyroyal, turpentine, and similar preparations may be used to keep off mosquitoes.

**Therapeutic.**—The management of a case of malaria is along general lines. During the chill warmth may be supplied or a hot bath may be given for relief. During the fever sponging with cool or tepid water, or a cool pack and an ice-cap to the head may be used. The convulsions sometimes seen should be treated symptomatically. The specific treatment is the administration of quinine. The dose should be relatively less than that for adults. Children, as a rule, bear quinine very well. Sometimes it may upset the stomach and in older children it may cause tinnitus. For very young infants the sulphate of quinine may be given in 0.03 gm. ( $\frac{1}{2}$  gr.) doses every three hours. At a year of age 0.06 gm. (1 gr.) may be given every two or three hours, and at two years 0.13 gm. (2 gr.) may be given. A child of six or over may be given much larger doses. The plan of giving a large dose several hours before the expected paroxysm is not of much use in young children, as it is likely to disturb the stomach. If even the smaller doses at regular intervals cause vomiting it may be given on an empty stomach at night, and then omitted during the day while the child is taking food. If it is persistently vomited twice the ordinary doses may be given by rectum, either in solution or suppository, or it may be given partly by mouth and partly by rectum. In pernicious cases, fortunately rare in children in this country, and in severe cases where quinine cannot be retained either by mouth or rectum, it may be given subcutaneously. This should never be done except when absolutely necessary, as it causes a great amount of local irritation and may cause sloughing and abscesses. The strictest aseptic precautions should be used.

The quinine should be kept up until the paroxysms cease and the temperature reaches normal, which it usually does promptly. After



smaller doses in the most palatable form should be administered for several days or a week.

In severe cases it is not well to trust to any of the various tasteless substitutes, but to use one of the salts of the alkaloid, as the sulphate or the bisulphate. For older children capsules may be used. Pills should not be given. For younger children the bisulphate, which is soluble in about ten parts of water, may be used. The taste is best disguised by using elixir glycyrrhizæ or the elixir eriodictyi aromaticum (verba santa), the syrup of orange or the syrup of sarsaparilla. The sulphate may be suspended in any of the above just before giving the dose. If it stands it will partially dissolve and cause a very bitter mixture.

For infants the plain solution of bisulphate and water usually answers best and is less apt to upset the stomach. In less severe cases and for use after an attack the less effective but more palatable forms may be recommended. There are syrups of cinchona, alkaloids, or so-called tasteless quinine. The dose varies with preparation; usually a teaspoonful represents 0.13 gm. (2 gr.) suspended in syrup. Euquinine, dose same as the sulphate, is tasteless and only slightly soluble in water; tannate of quinine, dose 0.06 to 0.9 gm. (1 to 15 gr.), insoluble and tasteless, generally given in chocolate tablets, which usually contain 0.065 gm. (1 gr.).

For rectal use the bisulphate or the sulphate may be made more soluble by means of tartaric acid; 0.16 gm. (2½ gr.) of the acid are used for each 0.9 gm. (15 gr.) of quinine. This dissolved in 2 teaspoonfuls of water and the proper amount added to barley-gruel is given as a high rectal injection. Rectal injections cannot be used very often, as the rectum becomes very irritable. They should ordinarily not be tried oftener than every six hours. Suppositories of quinine may be used. The hydrochlorate or other salt may be mixed with cocoa-butter; 3 drachms of cocoa-butter will make 12 infant-sized suppositories.

For hypodermic use the following is the formula of Bacelli, who also recommends this for intravenous injection:

R—Quinin. bismuriat.	1.00 gm.	(gr. xv)
Sodii chlorid.	0.06 "	(gr. j).
Aq. destil.	10.00 c.c.	(Sils) — M.

If this is not at hand the following may be substituted:

R—Quinin. bisulphat.	1.00 gm.	(gr. xv)
Acid. tartaric	0.15 "	(gr. iiss).
Aq. destil.	10.00 c.c.	(Sils) — M.

After an attack of malaria the child should be built up by use of tonics. Iron, quinine and strychnine are the best. Arsenic may be given in small doses, with good result. In the chronic cases or where there are constantly recurring attacks a change to a clear, invigorating climate will do more than drugs.

**EPIDEMIC CEREBROSPINAL MENINGITIS.**

By D. J. MCCARTHY, M.D.

This form of meningitis is due to a specific micro-organism—the diplococcus intracellularis meningitidis. It occurs in epidemics, but sporadic cases and localized epidemics, especially among children, are not infrequent. Children are more susceptible than adults. The effect of overexertion, excessive heat, and bad hygienic surroundings are important factors. The disease is not regarded as directly contagious, and is prevented in hospitals by simple antiseptic precautions on the part of nurses and doctors. Sporadic cases are not infrequently met with in large cities, and may occur in institutions for children with little tendency, apparently, to the production of an epidemic. Epidemics have been more frequently met with in the country than in cities.

**Pathology.**—The cause of the disease is an encapsulated diplococcus, easily stained by methylene blue, and found within the polynuclear leukocytes, although it may be free in the cerebrospinal fluid. In its morphological characteristics it resembles the gonococcus. The brain and spinal cord are both affected by the inflammatory process. In cases running a very rapid course the brain and spinal cord are very red, due to intense congestion. If the process lasts any length of time, a thick, pussy exudate forms at the base of the brain; in other cases the exudate is of a more fluid character and covers the entire brain and spinal cord. In the spinal canal, the lower portions of the cord, and especially the posterior surface, are more intensely affected. In chronic cases the exudate becomes organized and results in localized thickening of the cerebral meninges, with involvement of the cranial nerves.

The purulent process is not only localized to the meninges, but extends into the brain substance with the production of areas of hemorrhagic inflammation and miliary abscess formation. The microscopic examination does not differ essentially from that seen in other purulent inflammatory processes; the diplococcus is, however, found both in the exudate of the brain and of the spinal cord. The diplococcus may be found in the nasal or conjunctival secretion, and it has been assumed that infection takes place in this way. A pneumonia may complicate the disease and may be due to the pneumococcus or the diplococcus intracellularis; the complicating pneumonia may be either a croupous pneumonia or more frequently in children a bronchopneumonia. Leukocytosis is always present in this form of meningitis.

**Symptoms.**—The symptoms usually develop suddenly with vomiting, a chill or convulsion and high temperature. The fever runs as high as 104° to 105° F., the pulse is full and rapid, respiration is increased, and the child is very sick. There is evidence of intense headache, pains along the spine and in the back, with persistent projectile vomiting. In the beginning the restlessness and irritability are accentuated by

PLATE 211



FIGURE 1. (Continued) M. 1. 1. 1.

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2



**hypersensitiveness** to light and sound. In two or three days these **symptoms** progressively increase until the child becomes stuporous and then unconscious. A delirium very often supervenes, usually of an active type. The irritation at the base of the brain and of the spinal cord produces a stiffness of the muscles of the neck and of the back, with marked retraction of the head and a tendency to or actual opisthotonos. The irritation of the sensory roots gives rise to pain in the extremities and a hyperesthesia of the skin of the body so intense that the slightest touch causes great suffering. The irritation of the anterior motor roots of the spinal cord produces spasms of a chronic or a tonic type in the extremities and the face. The irritation of the motor nerves at the base of the brain produces strabismus, grinding movements of the jaws associated with dilatation of pupils, and the loss of reaction to light.

About the third day, but sometimes later than this, an eruption appears over the entire body. The typical eruption is a series of petechial spots immediately beneath the skin, which do not disappear on pressure. Other hemorrhages deeper in the tissues produce a purplish mottling here and there. In an epidemic which I recently studied an erythematous rash was present in a large number of the cases. In some cases there may be no eruption whatever. Herpes about the lips and sometimes elsewhere on the body is of frequent occurrence. The tache cérébrale is readily produced. The bowels may be either constipated or persistently loose. Toward the end of the week convulsions may develop, the pulse becomes very rapid, the active delirium subsides into that of the muttering type, and the patient dies from febrile exhaustion or cardiac failure.

In mild cases the fever is not so high and the irritative symptoms less intense. Irritability and restlessness, with rigidity of the muscles of the neck and of the back, intense headache, and slight delirium may be the only symptoms present; but even in the mild cases the auditory or optic nerve may be affected in such a way by the inflammatory process as to result in blindness or deafness.

Malignant cases sometimes occur, running a very rapid course and ending fatally in two or three days. The headache and intense pain are followed within twenty-four hours by a wild delirium and coma, convulsions, retraction of the head, opisthotonos, cardiac failure, and death. The fever may be very high, but more frequently is only of moderate elevation. The rash is usually purpuric in type.

The course of the disease is very variable, the temperature curve is irregular, and marked remissions in the symptoms may occur. A characteristic feature is the inequality of the pulse and temperature. The pulse is irregular and it may be low when the temperature is high.

Death may occur at any time during the disease from cardiac failure, pneumonia, arthritis, cystitis, or gangrenous bed-sores. In cases which go on to recovery (and cases exhibiting the most intense symptoms at times recover) an exhausted and asthenic condition persists for a long time. The patients remember nothing of what has happened

during the course of the disease, show little interest in what is taking place about them, often fail to recognize their closest relatives, and make little effort to talk or care for themselves. In some cases it may be several weeks or even months before an approach to the normal mental or physical condition is manifest. The sequelae are often very serious. Chronic hydrocephalus with marked mental deficiency may result in infants from the exudate at the base; even where this does not occur paroxysms of severe headache or of pain in the extremity may persist for a long time. Deafness often occurs and usually results in children in deaf-mutism. About one-fifth of all cases of deaf-mutism are occasioned by this disease. Blindness may follow a neuritis of the optic nerve or a septic process in the eye itself.

**Prognosis.**—The mortality varies greatly in different epidemics and may range from 20 to 75 per cent. In the sporadic cases prognosis should always be guarded, but can usually be made toward the end of the first week by the intensity of the symptoms presented at this time. A violent onset with convulsions generally indicates a severe type of infection and a fatal result may be expected. A rapidly developing coma early in the progress of the disease is of bad prognostic omen. A diminution of the leukocytosis, while not of definite prognostic import, may be regarded as favorable. The prognosis must, however, always be guarded.

**Diagnosis.**—When the disease occurs in epidemic form the diagnosis even of the mild cases is comparatively easy. There is, however, greater difficulty in the sporadic cases. The symptoms present do not differ essentially from those of other forms of meningitis. When, therefore, a case of meningitis occurs, for which no possible source of infection can be found, the possibility of epidemic cerebrospinal meningitis should always be considered. In typical cases where rash is present a positive diagnosis may be made from the symptoms alone. In all other cases it must depend upon an examination of the cerebrospinal fluid (p. 382). The fluid obtained may be perfectly clear in the earlier stages and in mild cases, or it may be cloudy and bloody purulent. Cover-slip preparations should be made and carefully studied by selective stains not only for the diplococcus intracellularis, but also for the diplococcus pneumoniae and the bacillus tuberculosis. When a differential diagnosis of tuberculous meningitis is in question the fluid may be injected into a guinea-pig to secure more accurate results. From typhoid fever the results of spinal puncture and Widal reaction are sufficient.

Recently, I was asked to see two cases, suspected to be cerebrospinal meningitis, in a large institution for orphan children. Both children had temperature ranging from 100° to 101° F., were very restless, irritable and paralyzed in the lower extremities. There was no evidence of headache, no retraction of the neck, no rigidity of the spinal muscles, no Kernig symptom, but there was distinct evidence of enlargement of the epiphyses of the long bones, a beginning rachitic rosary and profuse sweating about the head and neck. A diagnosis of p

paralysis, of rickets and scorbutus was made with a favorable prognosis. Both cases recovered from the acute symptoms after several weeks of treatment directed to the correction of diet.

**Treatment.** The general treatment is the same as that for other forms of meningitis, *i. e.*, symptomatic. While this form is not known to be definitely contagious, it is a wise precaution to isolate the patient in charge of a trained nurse. The room should be quiet and dark. The bowels must be kept open and the bladder must not be allowed to become distended. Irrigation of the nose and nasopharynx with normal salt solution will lessen the dryness of the membrane and wash away secretions of mucopus. Aufrecht and others have obtained good results from the use of the tub bath at 98° to 104° F., repeated as often as every three hours. Lumbar puncture apart from its diagnostic value may in some cases relieve pressure symptoms, and in this way produce good results. In recent epidemics the antitoxins used in other infections, and especially the antitoxin of diphtheria, have been much lauded as having curative properties. There is no reason, from a scientific standpoint, why they should have any effect. There is no reason, on the other hand, why they should not be used. Inasmuch as even severe cases of this disease recover under the symptomatic treatment, it is difficult to decide how much value there is in empirical serum therapy. A specific antitoxin for the diplococcus intracellularis should, theoretically, give good results in cases that have not advanced to destruction of nervous tissue. Trephining has been used in a few cases, but the results claimed for it can only be due to the relief of the pressure symptoms. Lysol and other agents have been injected into the dural canal without definite results. The treatment has been well summed up by Huber<sup>1</sup> as "necessarily empirical and symptomatic."

## INFLUENZA.

By MATTHIAS NICOLL, JR., M.D.

Pandemics of Influenza, or Grip (la Grippe), have been described for many centuries; the last severe one began in 1889, and spread quickly over the whole world. Following such an epidemic local outbreaks continue to appear at intervals for many years, occurring most frequently in the winter and spring.

The number of cases occurring among children varies in different epidemics. In some they are more frequently attacked than adults. At no age is there immunity to the disease. Children under one year are relatively immune, especially nurslings under six months, although influenza has been observed even in the newborn where mothers have contracted the disease before confinement.

**Bacteriology.**—The specific germ was described by Pfeiffer from observations made during epidemics above referred to. Certain pecu-

<sup>1</sup> Archives of Pediatrics, May, 1906, p. 33%.

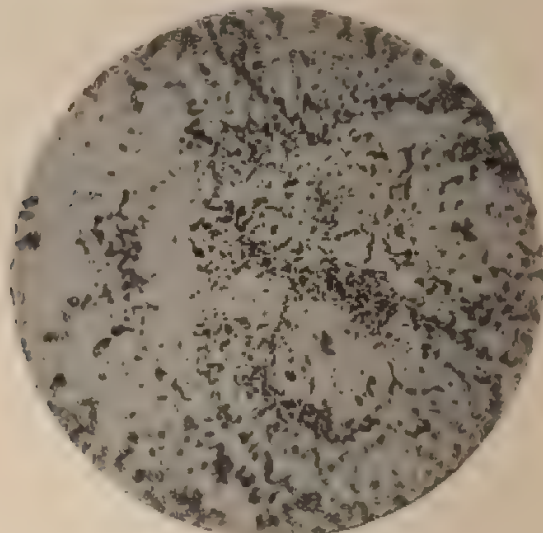


liarities of this organism should be known in order to understand the various symptoms of the disease and its mode of propagation.

It is a very short, small bacillus, or elongated coccus, found in great numbers in the bronchial and nasal secretions, especially at the early stages of the disease. It grows readily on various culture media to which whole blood and hemoglobin have been added after eighteen to thirty hours' growth at a temperature of 37° C. (98.6° F.). It forms small colonies represented by glistening points on the surface of the media, which show little tendency to coalesce (Figs. 102 and 103).

Stained with a 10 per cent. carbol-fuchsin solution the bacilli are seen as short, rather thick rods, varying greatly in dimensions and staining qualities, slightly rounded at both ends, and arranged in masses, or often in short threads. They apparently do not form spores, are killed at a temperature of 42° to 45° C. (107° to 113° F.) and below 3° C. (26° to 27° F.), and very rapidly by drying.

FIG. 102



Influenza bacilli.

Smears taken from the nose and bronchial sputum at the early stage of the disease show the bacilli in enormous numbers, at first free and not associated with other organisms to any great extent. Later they are within the pus cells, together with many cocci of the pyogenic variety and still later not at all. They have been found repeatedly in sputum of patients suffering from chronic pulmonary conditions, notably tuberculosis, for weeks and months apparently in a latent condition.

The influenza bacillus shows a greater tendency to associate with other organisms, especially pyogenic cocci, than any other disease, with the exception of measles and scarlet fever.

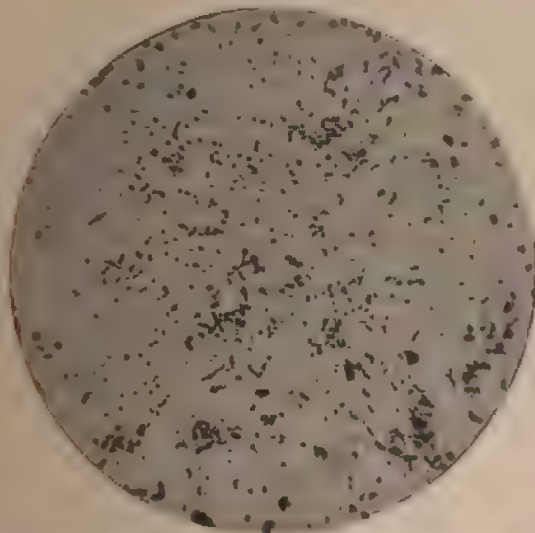


At the onset the symptoms of an attack of grip are attributable to the local action of the Pfeiffer bacillus and the toxins elaborated in its growth. Very soon, however, if the disease is protracted and complications occur, it is due to the action of the associated organisms and only indirectly to the grip bacillus.

**Ecology.**—Man, of all animals, appears most susceptible to the disease; indeed, it shows but little pathogenicity for most of the lower animals.

For man it is highly infectious; the germ apparently enters by way of the nose or mouth, and extends rapidly to the respiration passage down to the large and medium bronchi, and occasionally to the stomach and intestines. It is apparently transferred from one person to another by means of finely divided secretions containing the bacilli, expelled by coughing and sneezing, also by the use of towels, handkerchiefs, and fingers on which the bacilli have lodged in a moist state. The

FIG. 103



Influenza bacilli  $\times 1000$ . Eighteen-hour culture stained with carbol-fuchsin.

possibility of transmission by means of letters, books, and goods from a distance has been held responsible for the spread of the disease to widely separated localities. This, however, has not been substantiated, and from what has been said of the readiness with which they succumb to drying does not seem tenable. The bacillus shows little or no tendency to invade the blood or lymph stream.

Influenza is a disease whose symptoms are largely made up of complications caused by other organisms. During the prevalence of an epidemic its recognition is not difficult under ordinary circumstances. Sporadic cases are often mistaken for other pathogenic conditions, and

*vice versa*, the diagnosis of grip being all too commonly made in the case of ordinary colds accompanied by coryza, and obscure febrile conditions of many kinds. Bacteriological examination has not been frequently made as a matter of routine, and although the direct method of examination does not present any great difficulties, unless made at an early stage, it is apt to give negative results.

**Pathology.**—There seems to be no characteristic pathogenic change due to influenza. The mucous membranes affected show swelling and marked congestion and catarrhal inflammation; the neighboring lymphatic structures often show congestion and hyperplasia. The spleen may be involved and also Peyer's patches.

**Symptomatology.**—When the disease affects children above five years of age it differs very little from the well-known symptoms seen in adults. Younger children present certain peculiarities in symptoms, and the disease in infants is quite distinctive. The incubation is very irregular, varying from a few hours to a week or even longer. The invasion may be, and usually is, abrupt, or there may be symptoms of malaise, loss of appetite, and irritability for several days previous to the invasion.

The disease may be divided into several varieties, according to the prominence of different sets of symptoms.

The *febrile* form may or may not show a moderate coryza. It is characterized by the presence of fever, usually high, and marked toxemia. The disease may begin with a chill, rarely a convulsion; there is great prostration; older children complain of headache, pains in muscles and bones, vomiting is frequent, and there is complete anorexia. The fever may last from twenty-four to thirty hours, and rapidly subside, leaving the children weak and prostrated, or it may run an irregular course for several weeks. In severe cases so-called relapses may occur. There may be an eruption on the body during the height of the fever. As a rule this is an erythema, which may closely resemble scarlet fever, and less closely and less commonly measles. This is the type of disease seen in young infants.

The *catarrhal* form of influenza is that which is observed most frequently in older children. It differs little, if at all, in its symptoms from the same type as it occurs in adults. In ordinary cases the attack comes on suddenly, occasionally with a few hours or even days of indisposition. There is often vomiting or a chill with acute coryza, followed by rapid invasion of the trachea and larger bronchi. The conjunctivæ are reddened, sneezing and coughing are incessant; the pharynx shows more or less intense congestion, less commonly a membranous process, due to associated organisms. The larynx may be involved with symptoms of catarrhal croup. If a smear be made from the nasal secretions or sputum at this time, it will be found to be loaded with the grip bacilli. There is high fever, commonly headache, pains in the joints and muscles, prostration, a marked irritability, or somnolence. The pulse is rapid.

The physical signs are those of a bronchitis of the larger tubes, less often, in the early stage, of the medium and smaller bronchi; râles of

various kinds are scattered over the chest. The cervical lymph nodes may be enlarged. The fever may last only a day or two, when it falls rapidly, or it may continue for several weeks. The children are left weak, anemic, with little or no appetite, and a bronchial cough which may persist for a long time, with copious expectoration of thick, sticky mucus, or one of the many complications which are associated with this disease may prolong the illness or even cause it to terminate fatally.

The *gastroenteric* form of the disease may occur in connection with influenza affecting the membrane of the respiratory tract or as a separate disease. There is usually a rapid rise in temperature, with the symptoms that occur with fever, a thickly coated tongue, complete distaste for food, and more or less constant diarrhea and vomiting. In the severe form of this type of grip the fever continues for several weeks, the stomach and intestinal symptoms are grave, and the spleen may be enlarged. The patients are markedly apathetic, and the picture is difficult to distinguish from that of typhoid fever.

*Cerebral Influenza.*—Any variety of influenza in children old enough to describe their symptoms may, and usually does, show the effect of the toxin on the brain. The headache may be intense, the children markedly apathetic, less often irritable, the skin hyperesthetic, the reflexes increased; convulsions, except in infants, are rare. In addition to this there is a type of influenza which is hardly possible to distinguish clinically from cerebrospinal meningitis, save by a bacteriological examination and the shorter course of the disease. All the ordinary symptoms of meningitis may be present. True meningitis occurring in the course of influenza or following it will be regarded as a complication, since it is due to associated organisms, pyogenic cocci, and pneumococci, and not directly to the action of Pfeiffer's bacillus.

*Influenza in Young Infants.*—In the febrile form the symptoms are those of high fever, rapid pulse, and marked toxemia. There may be vomiting or the disease may be ushered in by a convulsion. The skin is often covered with sweat, and an eruption, generally an erythema, is not uncommonly observed. The infant shows absolute lack of desire for food, and usually lies in an apathetic condition. There is marked hyperesthesia, and from time to time the patient may cry out as though in pain. The symptoms may last only a day or two, when they disappear with a fall in temperature, or may last for several weeks, with increasing emaciation and weakness.

In the catarrhal form the picture is quite like that of measles complicated by pneumonia. There is congestion of the upper air passages, and later nasal discharge. The pulse is rapid, the temperature high, and the respiration is apt to be very much accelerated. The physical signs in the chest vary; they are usually slight compared to the severity of the symptoms. If present, they are those of a bronchitis of the larger tubes. The cough is persistent. These cases are frequently classed as abortive pneumonia to which the symptoms very closely correspond. The course is usually short, the temperature very often falling abruptly after twenty-four to forty-eight hours.



In the gastroenteric cases the symptoms are those of acute gastroenteric catarrh, aggravated by influenza toxemia. There is anorexia, a coated tongue, somnolence, prostration, and rapid emaciation. The vomiting is persistent, and the diarrhea may be of a severe type, with high fever and rapid pulse.

**Complications and Sequelæ.**—These are due in part to the toxemia produced by the Pfeiffer bacillus, but much more to the action of various organisms, notably the streptococcus, staphylococcus, and pneumococcus. In infants bronchopneumonia is the most frequent and dangerous complication. Certain characteristics of grip pneumonia have been described, but it is doubtful if they differ essentially from bronchopneumonia secondary to other infectious diseases. The type most commonly observed is that in which there are small areas of consolidation scattered throughout the greater part of both lungs with marked involvement of the bronchi, large and small. When larger areas are present there is frequently a complicating pleurisy.

Lobar pneumonia is not a usual complication of influenza in children. Severe bronchitis of the smaller tubes is to be regarded as a complication rather than a part of the disease. It is characterized by severity, persistence, and, in older children, the presence of profuse expectoration.

Suppurative processes, affecting the middle ear, not infrequently extending to the mastoid cells, of the accessory sinuses of the nose, of the cervical nodes, and occasionally of those back of the pharynx, leading to retropharyngeal abscess, are characteristic of certain epidemics. Nervous symptoms of various kinds, irritability, mental depression, neuritis, and neuralgia do not follow attacks of influenza in children with nearly so great frequency as in adults. Nephritis occasionally is seen and should not be neglected. In infants recovering from influenza, malnutrition and physical weakness may remain for a long time. Marasmus occasionally follows. Older children are left very often in a feeble state of health, with marked anemia, loss of appetite, and diminished muscular power; they not infrequently become the victims of tuberculosis.

**Diagnosis.**—This is not difficult during the prevalence of an epidemic, especially in the case of older children. In infants it often presents great difficulties. In general, it is based on the presence of the disease in the locality, on the disproportion between the local lesions and physical signs, and the severe clinical symptoms. The high fever, great prostration, rapid pulse, and other evidences of toxemia serve to distinguish catarrhal influenza from an ordinary cold and gastroenteric influenza from other gastroenteric disturbances.

Influenza pneumonia is characterized by the severity of the symptoms, the high fever and rapid pulse, absence of large areas of consolidation, and consequent absence of marked physical signs.

From measles and scarlet fever the differential diagnosis is based on the course of the respective diseases. From meningitis, cerebral grip cannot be differentiated except by waiting for the outcome of the disease, and possibly, in the cases of cerebrospinal and tuberculous meningitis, by examination of the spinal fluid withdrawn by lumbar puncture.



From typhoid fever the severe form of enteric influenza may usually be differentiated by the atypical temperature of the latter, its usually shorter course, absence of rose spots, and of Widal reaction. Finally, it is probable that a more frequent examination of smears from the nasal and pulmonary secretions will in many cases serve to render a probable diagnosis.

**Course of the Disease and Prognosis.**—Uncomplicated grip in children usually ends in recovery. Commonly after a short time with the patient left in the feeble condition just described. In infants the prognosis in uncomplicated cases is good, but pulmonary complications and gastro-enteric disturbances so frequently occur that the disease is in such cases a very serious one. Less often infants are overwhelmed by the toxemia of the disease itself.

**Treatment.**—There is no specific for influenza. Epidemics of the disease are regularly followed by epidemics of quack remedies. Each case must be treated according to the symptoms which arise.

The different salts of quinine are quite universally regarded as having a favorable action on the course of the disease. In older children they may be given in full doses provided the stomach is not upset by so doing. In infants they should not be given. Stimulants, especially whiskey and brandy, should be used whenever the condition of the pulse and evidence of great physical weakness require them. In infants they are often needed throughout the disease. To older children in order to reduce the fever and act as a nervous sedative, phenacetin 0.06 gm. (1 grain) to each year of the child's age up to 0.19 to 0.26 gm. (3 or 4 grains) and repeated at four-hour intervals if necessary; antipyrin 0.03 gm. ( $\frac{1}{2}$  grain) repeated are of advantage. The effect on the pulse should be carefully watched; hyperpyrexia should be treated by means of cold baths, sponges, or packs. As in most infectious diseases, a brisk cathartic is of advantage in the beginning. The gastroenteric form is to be managed in the same way as gastrointestinal attacks due to other causes. The bowels should be cleaned out with castor oil or calomel, abstinence from food for twelve hours or more should be enforced, and the substitution of carbohydrates for milk until the bowels become fairly normal, when the milk should be given much diluted and in small quantities or peptonized.

The treatment of the pulmonary complications is that of broncho-pneumonia and bronchitis in general. Cases of influenza should be isolated as carefully as possible from other members of the family. Infants especially should be guarded against exposure to the disease.

Suppurative conditions of the mastoid cells and accessory sinuses of the nose call for surgical interference. Some of the severe cases of headache which have followed grip have frequently been shown to be due to the latter condition and prompt relief has followed the evacuation of pus and drainage.

The after-treatment consists of the administration of tonics, especially iron and cod-liver oil and careful feeding. When these fail a change of climate will often prove successful in restoring the health.

## CHAPTER XVIII.

### WHOOPIING-COUGH—MUMPS—GLANDULAR FEVER.

#### WHOOPIING-COUGH.

By MATTHIAS NICOLL, Jr., M.D.

**WHOOPIING-COUGH**, or Pertussis, is an acute infectious disease characterized by a catarrh of the upper respiratory system, hyperesthesia of the mucous membrane, and more or less frequent paroxysms of violent cough, succeeded by a deep inspiration through a partly closed glottis, causing a peculiar "whoop" or "kink."

While there can be no doubt that the disease is due to a specific organism, yet notwithstanding many reports of its isolation there cannot be said, at the present writing, to be sufficient unanimity among the different observers as to its identity to justify the belief that the specific organism has yet been discovered.

**Mode of Infection.**—The disease is apparently transmitted from one person to another by means of the breath or the atomized secretions from the nose and mouth expelled by coughing and sneezing. Fairly close contact is essential for its transmission. Outside of the body the organism does not seem to possess great vitality; rooms which have been occupied by a whooping-cough patient are apparently free from the disease shortly after their vacation. Infection from the clothes, hands, etc., through a third person cannot be disputed, but very rarely occurs.

**Occurrence.**—Whooping-cough is endemic in all large cities; in country places and in small towns local epidemics occur from time to time. The simultaneous occurrence of measles has been frequently observed. There seems to be little difference in the season as regards the number of cases.

**Age.**—The general susceptibility to the disease is very great. At no period of life is there immunity. Children a few hours old have been attacked as well as adults far advanced in years. There is, however, a great difference in susceptibility at different ages. From 35 to 50 per cent. of cases occur in children under two years of age. Infants under six months and especially nurslings seem to possess a certain immunity, but whether this is actual or due to the fact of their being exposed less frequently is not definitely determined. After the second year the number of cases shows a marked falling off, and diminishes rapidly from the fifth to the tenth year, after which the disease is not common, undoubtedly due to the fact that most children over ten years of age

have been rendered immune by an attack earlier in life. Girls and boys contract the disease in about equal proportions.

**Contagion.**—Whooping-cough may be conveyed from the very beginning of catarrhal to the second or spasmodic state. Some observers believe that it is only infectious during the catarrhal period, basing their opinion on hospital statistics, where cases in the spasmodic stage brought to children's institutions failed to convey the disease in a single instance. Comby modifies this opinion, believing that the possibility of infection is much greater in the catarrhal stage, but that during the spasmodic stage also the disease may be transmitted, after which the probability of infection is remote.

**Pathological Anatomy.**—There may be said to be no distinctive pathological findings in uncomplicated whooping-cough. There is regularly found at autopsy in such cases more or less congestion and catarrhal inflammation of the upper air passages, especially about the larynx and within the trachea. The lungs quite regularly show more or less emphysema in prolonged and severe cases, especially at the anterior border and apices.

**Symptomatology. Course of the Disease.**—This is usually divided into three stages—catarrhal, spasmodic, and stage of decline.

**The Catarrhal Stage.**—After an incubation, which varies from a few days to two weeks, the child is attacked by what has every indication of being an ordinary cold. The eyes are moderately affected; there is a catarrhal rhinitis; the pharynx is congested. The children are not particularly ill. There may be slight lassitude and loss of appetite. There is a moderate rise in temperature and cough. Various characteristics have been attributed to this cough, and yet, in the great majority of cases, it cannot be said that they are sufficiently in evidence to lead one to suspect the nature of the disease. A paroxysmal cough at night is frequently observed at the beginning of the pertussis. The physical signs, if any are present, are those of a bronchitis of the larger tubes. The cough shows no evidence of amelioration, becoming more constant from day to day, and toward the end of the period taking on a paroxysmal character, even before the appearance of the "whoop." The duration of this stage is most variable; it is usually placed as two weeks; in some cases it continues throughout the disease; in others, especially in severe cases, it is very short, and the children appear to whoop from the beginning of the disease. In young infants the paroxysmal character of the cough may be present almost from the beginning, often without the characteristic whoop. Some of these cases are not easily detected early in the disease.

**Spasmodic Stage.**—This is characterized by the paroxysmal cough peculiar to the disease; the attack comes on suddenly. From adults and children old enough to describe their sensations it is learned that the premonitory symptoms are a tickling or sense of irritation in the larynx, producing an uncontrollable desire to cough, and as the spasm of the glottis occurs there is a sense of dread of impending suffocation, which the late Dr. O'Dwyer, having himself contracted the disease in



adult life, describes as appalling, and as "though his very last second had come."

The attack begins with a series of explosive expiratory efforts following one another in rapid succession, no inspiration being taken between them; then a deep inspiration through the partly closed glottis, accompanied by the characteristic whoop. After a very short time the phenomenon is repeated; frequently several times, until finally a plug of mucus is expelled, when the attack terminates, often with vomiting.

At the approach of a paroxysm the child stops in its play, runs to someone as though for relief from the dreaded sense of suffocation. Later in the disease, when it has become more or less accustomed to the attacks, it seizes a chair, table, or side of the bed as a support during the paroxysm. While the explosive expirations are taking place, the face and head become red or dusky, the conjunctivæ congested; the eyes water and appear to bulge from the socket; the nose runs, and the whole body is drawn into a state of spasm, and covered with sweat; the pulse is very rapid. With the occurrence of the final whoop all the muscles are relaxed and the child remains quiet, in a state of complete exhaustion. The number of paroxysms occurring in twenty-four hours varies from a dozen to eighty or a hundred.

Many theories have been advanced to explain the exciting cause of the paroxysm. It is generally attributed, and with good reason, to the plug of mucus in the larynx or trachea, presumably containing the infectious organism. After this has been expelled the attack ceases.

During this stage there may be a moderate rise in temperature, or the disease may run an afebrile course. Its duration is variable, from two weeks to two months or more. With every fresh cold the spasmodic attacks may be repeated, often after several months, due apparently to the hyperæsthetic state of the air passages rather than to a true relapse.

*Period of Decline.*—The spasmodic attacks of the second stage, having grown less and less frequent, the whoop at last disappears, and the disease enters the third and final stage. This is characterized by a cough, having at first something of a paroxysmal character, and gradually assuming that of ordinary tracheobronchitis, which continues for two or three weeks and ceases. If for a longer period it is due, as a rule, to complicating general bronchitis, or other pulmonary lesions.

Variations in the usual type of the disease occur. The course of the disease may be very prolonged, even without evidence of complication or it may run a very short course of a week or more. A cough at the first stage may disappear, and then suddenly the spasmodic stage be entered into. The disease may resemble a stubborn bronchial cough from the first, its true character only being recognized by the existence of whooping-cough in other members of the family, with characteristic symptoms, or after the disease has lasted for some weeks.

General symptoms in uncomplicated cases besides those mentioned are the occurrence of leukocytosis, which may be very marked. Albumin occurs in small quantities in about half the cases, occasionally accompanied by casts.



PLATE XIII.



Subconjunctival Ecchymosis in Whooping-cough.



**Complications and Sequelæ.**—The complications of whooping-cough may be divided into those caused by the mechanical effect of the spasmodic cough and those due to infection by various organisms. To the first class belongs the ulcer, covered with gray-white membrane, seen on the frenum of the tongue, or just in front of it, and caused by the pressure of the latter on the lower incisors during a paroxysm. While it is fairly characteristic of the disease it occurs in other forms of severe cough.

Emphysema, as already stated, regularly occurs. It is usually moderate in extent and seen at the apex and anterior borders of the lungs. It may rarely be of a severe type, with rupture of the lung, the formation of pneumothorax, and general subcutaneous emphysema. According to O'Dwyer, emphysema is due to the recoil of air against the wall of the pulmonary vesicle during the forced expiratory efforts through an almost closed glottis. Furthermore, after the lung has been all but emptied of air, that which remains expands, causing a partial vacuum, which the subsequent inspiratory effort through a partly closed glottis fails to fill, but, on the contrary, the expansion in the chest tends to increase; so that, according to this view, both expiration and inspiration take part in the production of this lesion.

The increase in the venous pressure during an attack leads very frequently to hemorrhage, which occurs during or following a paroxysm and takes place most frequently from the nose, mouth, or trachea. Hemorrhages of the conjunctiva are not uncommon and give a very characteristic picture. In addition, the cellular tissue beneath the eyes may be the seat of hemorrhage with the appearance of "black eyes." Hemorrhages from the ears have been frequently described; the drum membrane may be ruptured during an attack, but usually this accident occurs in a middle ear already affected by an antecedent disease. (See Plate XIII.)

Hemorrhages into the brain and pia mater are usually small, frequently multiple, less often large; they lead to various paralyses, disturbances of equilibrium, and mental symptoms, depending on their location. Dilatation of the right heart to a greater or less degree is not uncommon; severe dilatation with relative insufficiency of the valves has been noted.

Hernias may be caused or increased, and prolapse of the rectum is sometimes noted with or without the existence of disease of the lower bowel.

Vomiting occurs as a regular symptom, and may be regarded as a mechanical result of the cough unless it be prolonged beyond the spasmodic stage.

Convulsions, usually seen in young infants, are due to intense cerebral congestion during an attack or to intracranial hemorrhage. Asphyxia may follow a severe paroxysm, with or without convulsions.

The second class of complications comprises those that are caused by infection, the most important of which is bronchopneumonia, which occurs much more frequently in infants than in older children, and especially those in the first year of life. It is much more often seen in

hospital cases and in tenement-house practice than amid favorable surroundings; more often in the winter and spring than the summer months. It comes on usually when the disease is at its height, in some part of the second stage. The onset may be sudden, or there may be symptoms for a number of days previously of a general bronchitis. The lesions consist usually of small areas of pneumonia scattered over a greater part of both chests; or there may be one or more large areas of consolidation. The respiration is usually very rapid and out of proportion to the temperature, due to the presence, perhaps, of a complicating emphysema. The whoop, if it has been present, often disappears, but the paroxysmal character of the cough usually remains. The disease is very fatal and is usually prolonged even if it terminates favorably. Convulsions frequently occur during the course of the pneumonia, or as a final symptom. Pleurisy frequently complicates the more chronic cases.

Bronchitis is a frequent complication in young children, often prolonging the third stage of the disease.

Otitis media and mastoid abscess are occasionally seen.

In summer-time the disease in infants is frequently complicated by severe diarrhea, which greatly adds to the gravity of the case.

Vomiting, instead of occurring only with the attacks of coughing, may be almost incessant and continue far into the stage of decline. Finally, any of the infectious exanthemata may and frequently do complicate whooping-cough, especially in institutions: measles often, diphtheria and scarlet fever not infrequently. Such a complication is very apt to cause a fatal termination.

Whooping-cough is not infrequently followed by general tuberculosis, the disease either lighting up a latent process usually in the bronchial lymph nodes, or being engrafted on a weakened constitution.

Marasmus occasionally follows severe cases in infants.

**Diagnosis.** This is generally impossible until the stage of spasm. One may suspect the nature of the disease when a bronchial cold without any or only very limited physical signs grows worse from day to day in spite of treatment.

In abortive cases and those without a whoop the diagnosis must be made on the other characteristics of the cough, history of exposure, and absence of physical signs.

Some children whoop to a moderate extent whenever they contract cold, but the character of the cough is not typical and the course of the disease is quite different from that of whooping-cough. Such children frequently have adenoid growths or a thickened pharynx.

Foreign bodies in the larynx have occasionally simulated this disease and led to a false diagnosis.

Enlarged bronchial lymph nodes pressing on the pneumogastric nerve give rise to symptoms in some cases hardly to be differentiated from those of whooping-cough. The course of the two diseases, method of onset, history of exposure, must be taken into account in determining the nature of the case.



**Prognosis.**—The aggregate mortality from whooping-cough is large, as the following statistics quoted by Comby<sup>1</sup> will show. In the city of Paris, from 1880 to 1900, 7613 deaths occurred from the disease. In the city of London in 1893, 2330 deaths. According to Johnston,<sup>2</sup> whooping-cough in the United States is responsible for the deaths of 100,000 children in every decade.

It is a much more fatal disease in institutions and in poor surroundings than when occurring under opposite conditions.

Age is a most important factor in determining the outcome. In children under two years, and especially those under one year, it is very fatal on account of the occurrence of complications, especially of pneumonia and convulsions. After two years the death rate gradually decreases, and after five it is very low. The prognosis is not as good in winter as in summer or in rachitic and debilitated children as in those previously in good health. It is not good when constant vomiting interferes with the patient's nutrition or when the disease is complicated by one of the exanthemata.

The severity of any uncomplicated case is to be judged by the number of paroxysms occurring in twenty-four hours, together with the violence and duration of the individual seizures.

**Treatment.** The number of remedies suggested for whooping-cough bears eloquent testimony to the lack of success attending any one kind of treatment, and yet a great deal can be accomplished in alleviating the sufferings of the patient, even though the course of the disease is not altered.

**General Measures.**—When the nature of the disease has been determined the child should be isolated in so far as possible from susceptible individuals and especially from young infants. The food should be easy of digestion, peptonized if necessary. The children may be required to be fed at frequent intervals if the vomiting is constant. Milk forms a suitable diet in many cases. In artificially fed infants it may have to be weakened or predigested.

There can be no doubt that these patients cough much less when out-of-doors than when confined to a closed room, for which reason they should be allowed out on good days as much as possible, the room being thoroughly aired and cleaned before their return. In inclement weather, fresh airings may be substituted. If it is necessary to confine the child to one or two rooms they should be aired and cleaned constantly. Frequent change of bed-clothes and wearing apparel are helpful. In severe cases which do not yield to ordinary measures, a change to a warm climate, preferably by the open sea, or a sea voyage, is often of great benefit.

The milder attacks of whooping-cough, especially when occurring in children over two years of age, require no other treatment than careful feeding, proper clothing, and fresh air.

<sup>1</sup> Traité des maladies de l'enfant

<sup>2</sup> Medical Society of the District of Columbia. January 23, 1895.

No remedy has yet been discovered which has a uniform effect in shortening the disease, but many diminish the number and severity of the spasms in a certain proportion of cases. A few of those which have been found most efficacious are as follows:

*Local Treatment.*—This consists of: 1. Insufflation of various powders into the nose and larynx; quinine mixed with some bland powder, as bicarbonate of soda or acacia, in the proportion of 1:10 or stronger; antipyrin, boric acid, and benzoin, the treatment being given three or four times a day, preferably just after a spasm. This method of procedure is at present but little in vogue. Its results are far from convincing.

2. Applications to the larynx, especially of cocaine solutions, 1 to 4 per cent., is undoubtedly efficacious, but it is a difficult method of treatment and the danger of poisoning must be borne in mind. Weak solutions of 1:60 and 1:90 of carbolic acid and other antiseptics may be also used in this way.

3. Inhalations of carbolic acid, creosote, and cresolin have been found of decided benefit. The air of the room may be saturated with one of these substances by means of a croup kettle or special apparatus made for this purpose, or clothes soaked in carbolic acid may be hung over the children's bed, or the substances used in an inhaler. The possibility of carbolic acid poisoning is to be guarded against by regular examination of the urine.

The treatment suggested by Bergson in 1887, and later used with marked success by Dr. O'Dwyer in the New York Foundling Hospital in the treatment of 150 cases of whooping-cough, consists of the rectal administration of carbonic acid gas. For this purpose there is needed a wide-mouthed bottle holding a pint or more, into which passes a glass tube through a perforated cork. A rectal tube is fitted to this with a nozzle suitable for introducing into the rectum. The bottle is filled one-third with water and 24 gm. (6 dr.) of bicarbonate of soda dissolved in it, after which 15.50 gm. ( $\frac{1}{2}$  oz.) of crystalline tartaric acid is added and the rectal tube inserted.  $\text{CO}_2$  is thus liberated in proper quantities. The treatment is continued for five to ten minutes, depending on the child's age, and given three times a day. A flushing of the face regularly follows the administration of the gas. The paroxysms are reduced in number and the whoop often promptly disappears, together with the vomiting. Occasionally the treatment will have to be suspended on account of the occurrence of a mild diarrhoea. While I have had no personal experience with this form of treatment, the fact that it has been vouched for by so good an observer justifies its further use, especially as it produces no dangerous symptoms. The breathing of the gas-laden air of gas tanks is in all probability a popular mode of applying this treatment.

For the immediate relief of threatened suffocation, or, in order to abort a spasm, pulling the jaw forward, as suggested by Nacgeli and lately by Sobel, is effective in a certain number of cases. The administration of chloroform or ether or the inhalations of oxygen is occa-

sionally indicated. Finally, intubation has been practised with more or less success, in this country and Europe, in desperate cases.

**Internal Treatment.**—Among the large number of remedies suggested the following have stood the test of time and experience as being efficacious in a good proportion of cases. Antipyrin, well borne by even young infants in doses of 0.06 gm. (1 gr.) to each year of a child's life up to 0.2 gm. (3 gr.), every two hours. The addition of sodium bromide in quantities double that of antipyrin has seemed to me to increase the efficacy of this method of treatment. In older children quinine in doses of 0.13 to 0.19 gm. (2 or 3 gr.) of the sulphate or its equivalent, three times a day, is well thought of by many. It may be given in syrup of yerba santa. When it upsets the digestion, which it is apt to do, it should be discontinued, and on no account should it be given to infants.

Belladonna, pushed to the point of tolerance, undoubtedly has an effect on the symptoms. Its routine employment is more or less limited to hospital cases and those which are constantly under skilled observation. The fluid extract may be given in doses of 0.032 c.c. ( $\frac{1}{2}$  min.) every four hours and gradually increased, or the intervals shortened. Flushing of the skin and dilatation of the pupils must be induced in order to secure the benefits of this drug.

A remedy suggested to me by Dr. Silver, of the Vanderbilt Clinic, in New York, and employed very frequently since, has produced good results in a majority of the cases in which it was used, namely, the internal administration of peroxide of hydrogen (3 per cent.), as in the following prescription:

Rx.—Hydrogen peroxid. . . . .	60.0 c.c.	(3ij).
Glycerinum . . . . .	15.0 "	(ss).
Aque . . . . .	q. s. ad 120.0 "	(1iv).—M

Sig.—Teaspoonful in water every two hours.

No bad results have followed this treatment, and the number of paroxysms and their severity have been regularly reduced.

Bromoform may be mentioned as having had many advocates at one time; poisoning has been reported in a number of instances. It may be given in doses of 0.18 to 0.30 c.c. (3 to 5 min.) three times a day, or more frequently, the initial dose being about 0.06 c.c. (1 min.) for a child of one year. It should never be given in a mixture that is allowed to stand or that is not well shaken.

**Treatment of Complications.**—Vomiting which persists after the spasmodic stage may require the temporary employment of rectal feeding or stomach washing, in addition to the use of readily digested food in small quantities at frequent intervals. For diarrhea and intestinal catarrh the usual treatment is to be employed: cathartics at the outset, castor oil or calomel, stopping the milk, and the substitution of carbohydrates, broths, soups, and chopped meat, together with lavage of the bowels when necessary.

Hemorrhage cannot be prevented except by control of the paroxysms. In severe epistaxis astringents may be tried, as douches or the local



application of adrenalin, 1:5000 to 1:10,000 solution, and, as a last resort, plugging the posterior nares.

Stimulants are needed in cases of weak heart and in most infants.

Bronchopneumonia complicating whooping-cough is to be treated in the same way as pneumonia complicating other infectious diseases, but needs very careful oversight because of the paroxysmal cough.

In institutions for children when diphtheria prevails immunizing doses of antitoxin should be given at the onset of the disease.

The after-treatment consists of cod-liver oil, syrup of the iodide of iron, etc., attention to the diet and general health, and, when possible, change of climate.

### MUMPS.

By MATTHIAS NICOLL, Jr., M.D.

Mumps (Infectious Parotitis) is an acute communicable disease characterized by swelling of the salivary glands and tendency to involve other glandular structures, notably those of the genital tract.

**Occurrence.**—The disease in children occurs most frequently between the age of five and fifteen years, though it is occasionally met with at a much earlier period. It occurs in small epidemics, especially in schools and other institutions where older children congregate. At the New York Foundling Hospital, in which nearly all the children are under five years of age, the disease is practically unknown.

**Etiology.**—Close contact is necessary for its dissemination, which is probably through the agency of the breath. It may rarely be conveyed by a third person, clothes, and other articles.

It is apparently contagious from the very beginning of the symptom to the time of subsidence of the glandular swelling, and even later.

One attack usually renders a patient immune during the rest of life, but second attacks are not very rare and relapses occasionally occur.

**Incubation.**—Cases have been reported as occurring one week, or even less, after exposure. The usual period of incubation is a long one, usually from eighteen days to three weeks or more.

**Bacteriology.**—A diplococcus isolated by Laveran and Catrin from the blood, testicles, and serous effusions occurring in the course of the disease has been identified by other observers, and while much evidence points to it as the specific cause, thus far, to my knowledge, no one has succeeded in reproducing the disease by inoculating cultures of the organism.

**Pathology.**—In the rare cases which have been brought to autopsy there has been found congestion of the gland involved, with catarrh of the tubules and edema of the surrounding connective tissues. In complicating orchitis, evidence of atrophy of the seminiferous tubule has been described.

**Symptomatology.**—In many cases there are no prodromata, or these are so slight that the first symptom noted is the swelling of the parotid.



gland. There is usually a rise in the temperature of two or three degrees.

In other cases the fever is high and accompanied by headache, pain in the back and muscles. There is loss of appetite and general languor. There may be chilly feelings or an actual chill. Cases of delirium have been described. The more violent method of onset is much more commonly seen in adults than in children. Other symptoms frequently complained of are sore throat and earache. Epistaxis may usher in an attack.

The swelling of the gland begins under the lobe of the ear, between the mastoid process and the ramus of the jaw, and extends upward in front of the ear and downward to the neck (Fig. 104). The swelling, when moderate, can best be appreciated by looking at the patient from behind

FIG. 104



Mumps.

in a good light. On palpation the swollen area is felt to be smooth, slightly resistant, and more or less sensitive to pressure. Both glands may be involved simultaneously, but in the majority of cases one precedes the other by two or three days or there may be a delay of a week or more.

There is pain on opening the jaw widely, and moderate soreness of the throat on swallowing. The patients are apt to speak and take food through a partly opened mouth; the flow of saliva is regularly diminished; it may be normal or even increased.

On examination of the throat, at the beginning of the disease, there is often seen a redness of the fauces, soft palate, tonsils, and of the inner surface of the cheek, together with a swelling of the mucous membrane at the orifice of Steno's duct.

Instead of the swelling being confined to the immediate vicinity of the parotid, it may extend upward to the orbital region and downward over the face and neck. When this swelling is bilateral the patients present a squirrel-like appearance. In severe cases the skin over the affected parotid may be reddened to a moderate extent, in which case the tenderness on pressure is quite marked.

The disease may not be confined to the parotid, but involve the submaxillary and sublingual glands, or one or both of the latter may be swollen without the parotid or may precede the swelling of the latter.

The swelling having reached its height gradually subsides, and the appearance of the gland is normal usually after a week to ten days. Relapses may occur.

Even when the disease begins with severe symptoms, after the first twenty-four hours the children are not particularly ill, complain only of a sense of discomfort and the difficulty in swallowing, especially, solid food.

**Complications.**—Mumps is a disease which in childhood, almost without exception, runs a benign course. The complications so frequently observed in adults are practically never seen, and the few complicated cases described have nearly all occurred at or near the age of puberty. Orchitis is occasionally seen. At its onset there is often high fever, restlessness, and even a mild delirium, together with pain referred to the testicle and cord, with some tenderness on pressure, or the symptoms may be so slight that only an examination of the testicle may reveal the fact of its involvement. The duration of this complication is usually from three or four days to a week or more, when complete resolution takes place. Atrophy is almost unknown. Orchitis may occur at the height of the parotid swelling, but usually during convalescence. Swelling of the mammary gland, not confined to girls, is occasionally seen, even at an early age. Swelling of the ovaries, with pain and tenderness on deep pressure, and of the external genitals in girls is sometimes met with.

Less common complications are: secondary involvement of the lymph nodes; suppuration or gangrene of the affected salivary gland, due to secondary infection; involvement of the thyroid and lacrymal glands; paralysis of the various nerves, especially of the auditory nerve, which may be followed by total and permanent deafness; paralysis of the facial nerve, apparently due to pressure, and nephritis.

**Prognosis.**—Mumps in children is a self-limited and rarely complicated disease, running a benign course. It is never fatal.

**Treatment.**—Cases occurring in schools should be isolated. Strict isolation within the house is not necessary. With the onset of the disease, the child should be put to bed and confined to the house during the entire illness. Sedatives may be required at the beginning, and cold sponges to reduce the fever. The diet should be fluid, preferably milk. To relieve the pain and discomfort, hot applications, camphorated oil, or belladonna ointment may be applied to the swollen gland.

Mild solutions of boric acid, listerine, or other mild antiseptic mouth-wash should be used several times a day. When orchitis occurs, rest in bed is immediately indicated, with the application of cold to the affected part. Guaiacol ointment, 25 per cent., as an inunction has been found efficacious in the treatment of this complication. Fumigation is not necessary except in schools and other places where a number of cases have occurred. Under such circumstances a thorough cleaning and application of antiseptics may be necessary to prevent further outbreaks of the disease. Three weeks from the beginning of the attack may be taken as the probable limit of the infectious period.

### GLANDULAR FEVER.

By FLOYD M. CRANDALL, M.D.

In 1889 E. Pfeiffer described a condition which he called Glandular Fever. He described two forms of the disease, the one very rapid in its course, the other less rapid, but still an acute disorder. Since that time numerous cases denominated glandular fever have been reported, but there is grave doubt as to the actual character of many of them. Some have unquestionably been only influenza or coryza, with enlargement of the lymph nodes. Others have been septic cases, and still others have been atypical cases of typhoid fever or other infectious diseases. The mere presence of the enlargement of lymph nodes with febrile symptoms does not warrant the diagnosis of glandular fever. It is my own belief that there is such a disease as glandular fever, but that it is very uncommon and rarely occurs sporadically. Pfeiffer asserts that it occurs usually in epidemics, but they are of limited nature. All the children suffer from it when it is introduced into a family. The best work in this country on this disease has been done by West, Hamill, and Seibert, and in England by Dawson Williams.

The most extensive and complete observations on glandular fever are those of J. Park West, of Bellaire, Ohio, reported in *Archives of Pediatrics*, December, 1896. He reports 96 cases observed by himself and Dr. Korell, occurring in Eastern Ohio among children between the ages of seven months and thirteen years, in forty-three families. Only twenty children of these ages escaped, but there were numerous children, both younger and older, who did not contract it.

**Symptomatology.**—The disease described presented the following train of symptoms: A sudden definite onset after a period of incubation; a fever, running its course in from four to seven days and terminating by crisis; characteristic enlargement of the cervical lymph nodes, forming an elongated tumor, lying below the angle of the jaw anterior to the sternomastoid muscle, beginning always on one side and appearing later on the other; enlargement in most cases of other lymph nodes, notably the postcervical, axillary, and inguinal, and not infrequently the mesenteric and bronchial; enlargement of the liver and spleen in a large proportion

of cases; prostration and rapidly developing anemia. The disease as recorded by West was clearly contagious, and occurred chiefly between the ages of one and ten years. Debility and a weak, rapid pulse were always present, and were noticeable in most cases after all other traces of the disease, except some swelling, had disappeared. The skin had a dull, flushed appearance, but there was no eruption of any kind. The eyes were heavy and frequently the pupils were widely dilated.

The most marked feature in all the cases reported by West was the enlargement of the cervical or, to be more definite, the carotid lymph nodes. After two to three days of malaise the swelling could be seen. As a rule it began on the left side and reached full development on the second to fourth day. Several hours before its completion on this side it would be noticed on the right, and the same course would be followed as on the left. Occasionally the swelling began on the right side, but in no case did it appear simultaneously on both sides. Very rarely was it confined to but one side. The swelling always had the same peculiar characteristic appearance. To the eye it was smooth, but the finger easily detected three or four enlarged lymph nodes. This swelling was elongated, about as thick as the index finger, and ran downward and forward from just below the angle of the jaw, between the body of this bone and the sternomastoid muscle, to a little beyond the middle of the jaw.

Other lymph nodes in the immediate vicinity were swollen, but not so much. The swelling was always tender, often painful, and frequently caused stiffness of the neck, and a choking sensation. In three-fourths of the cases there was noticed enlargement of the other lymph nodes—postcervical, axillary, and inguinal. They were never all enlarged in any single case, nor were they so much enlarged nor so tender as the cervical nodes. In thirty-seven cases the mesenteric lymph nodes could be felt enlarged. This is probably considerably understated, as examination was not made in the earlier cases.

The history of the cases in this epidemic resembles very closely that given by Pfeiffer in his second class, and bears out his statement that the disease is of epidemic character that does not extend beyond the children of a single house or family. So far as is known only one adult case has been described. The disease is usually at its height on the third or fourth day, at which time the temperature reaches its maximum point. The acute symptoms subside rapidly, but convalescence is apt to be slow and tedious. The disease is rarely, if ever, fatal. The most serious and frequent complication is acute nephritis, ten cases of which have been recorded in literature.

**Diagnosis.**—There are some who doubt the existence of glandular fever. Ashby and Wright are "rather inclined to think that while 'gland fever' does undoubtedly occur, it is rarely idiopathic, but the results of absorption of toxic materials from a mucous membrane." Several writers mention it as a result of autointoxication, with the intestinal tract as the probable source of the infectious material. Others are of the opinion that there is a probable microbic influence, while



## CHAPTER XIX.

### SCARLET FEVER.

By FLOYD M. CRANDALL, M.D.

SCARLET fever, or Scarlatina, is an acute, infectious, and contagious disease, occurring commonly during childhood. Typical cases present the following features: After an incubation of from three to four days there is a sudden onset of sore throat, vomiting, and fever, followed within twenty-four hours by a rash, consisting of minute points of a scarlet color closely grouped on a reddened skin, which appears first on the neck and extends rapidly over the body. The eruption continues from four to six days and is followed by a stage of desquamation which continues from three to six weeks. The disease may be contagious from the first symptoms, but is usually not contagious until the rash has appeared. The period of contagion continues until desquamation is complete.

**Etiology. Exciting Causes.**—Scarlet fever is beyond all doubt an infectious disease, but the specific germ has not yet been discovered. It seems certain that *streptococci* play an important role in the causation of some of the symptoms, but the evidence seems to be growing stronger that streptococci are not the cause of the disease itself. The recent studies of Hektoen, Weaver, and Ruedinger strengthen the idea that the streptococcus is an important factor in making up the symptom complex of scarlet fever, but lend no support to the claim that it is the specific organism. Hektoen points out that while streptococci may be found in the blood and internal organs after death, they are sought in vain in the early stages and are absent in the majority of cases until late in the disease. The significance of the fact that streptococci are largely found after death is lessened by the other fact that in many conditions like measles, diphtheria, and smallpox the same organisms are frequently found. The most reasonable assumption at present is that in scarlet fever we have usually a mixed infection by the streptococcus and a yet unknown specific germ.

Weaver asserts that streptococci obtained from the throat of scarlatina patients are not different in structural, cultural, and morphological peculiarities from the streptococci obtained from other sources. Baginsky found scarlatinal blood serum to have no agglutinating action upon streptococci, but Moser has produced a serum which agglutinates streptococci from scarlatinal cases in a different manner from other streptococci. Weaver and Ruedinger also failed to find any agglutinating

fection. These observations seem to strengthen the idea that the streptococcus obtained so often from the bodies of those who have died from scarlet fever is nothing but the common streptococcus and not the specific micro-organism of scarlet fever.

Some recent observations have been made which show that the severity of the disease is in direct proportion to the streptococcemia. Among the cases designated as mild, streptococci were found during the first week in but 9 per cent., while in those designated severe they were found in 27 per cent. Whatever the cause of the primary disease may be proved in the future to be, it is certain that streptococci are the cause of some of the secondary symptoms, and must be regarded as important factors in the production of the usual clinical picture which we know as scarlet fever. Staphylococci and diphtheria bacilli are sometimes found in conjunction with the streptococci. These germs were present in a recent case of my own.

It seems certain that the specific germ of scarlet fever exists in the blood, for inoculation of the serum into susceptible animals produces a typical attack of the disease. It must also exist in the secretions of the mucous membranes, in the desquamation scales, and possibly in the keratins, as shown by their power to generate the disease. Some of these questions cannot be settled definitely until the specific micro-organism is found.

*Predisposing Causes.* Among the predisposing causes age must be placed first. The disease is rare under one year, but I have seen an undoubted attack of scarlet fever in an infant of one week. It should not be forgotten that albumin is sometimes found in the urine during the first days of life, its presence then being of little significance. After the first week its occurrence is of the same significance as later in life. It is also to be remembered that hyaline casts may frequently be found in the urine of perfectly healthy infants during the first week of life. Granular casts are also found, but are less common than the hyaline. It is thus evident that during the first week or ten days of life urinary analysis may prove very misleading if judged by the adult standard, and the presence of albumin and casts may not indicate an infectious disease. Up to five years the susceptibility to the disease steadily increases and reaches its maximum; after eight years it rapidly decreases, and is slight during adult life. Sex does not influence its occurrence.

Scarlet fever is a far less common disease than is measles and susceptibility to it seems to be much less. While almost every child who has not already had measles may be expected to contract it upon exposure, at least half the children exposed to scarlet fever may be expected to escape unless the exposure is close and prolonged. Epidemics of scarlet fever are usually less frequent than those of measles and are rarely as widespread. Epidemics are most common during the fall and winter months. Several observers have found it to be more common during October than in any other month of the year, and the mortality higher. Epidemics of scarlet fever usually spread very slowly as compared with those of measles.

*Sources of Infection.*—The chief source of infection is the patient himself, but the area of contagion is limited to a few feet. The desquamation scales are extremely infectious. Their retention by clothing, bedding, and the walls of the rooms is one of the most common causes of infection. The purulent secretions from the throat, nose, and ear are also very infectious. Scarlet fever is spread by indirect infection more frequently than any other disease except smallpox. Its specific micro-organism is more tenacious of life than that of any other disease except perhaps smallpox. It may be conveyed from one child to another in the fur of cats and dogs, and it is probable that these animals may suffer from the disease. The contagion clings to rooms with great tenacity, being frequently lodged in the wall-paper or in cracks of the walls, ceilings, and floors. The conveyance of scarlet fever by milk and other articles of food is undoubted. The celebrated epidemics of Hendon and Wimbledon were believed by Dr. Klein to be due to scarlet fever in the cows, but this belief has not been substantiated. It is probable that the disease from which those cows suffered was not true scarlet fever.

The disease has been conveyed by letters written by hands in the stage of desquamation. An attendant upon a case of scarlet fever may easily carry the infection to other children by the clothes, hands, or beard. Such transmission is probably not common, however, except when the contact has been close and somewhat prolonged. The clothing of a nurse which comes in close contact with a patient for extended periods of time may be highly infectious. It is certain that the greatest danger of infection lies in the transmission of the desquamation scales. Holt asserts that in a city the bed-clothing while airing in a window has been known to convey the disease to an adjoining house, and records also the same result from the washing of infected with other clothes. It would scarcely seem possible that scarlet fever could be conveyed through two healthy persons, but a few apparently authentic cases of this kind have been recorded. This would apparently result from the transmission from person to person of desquamation scales.

*Portal of Entrance.*—The portal of entrance is undoubtedly in most cases the nasopharynx. It is here that the first local symptoms appear, and the secondary micro-organisms at least commonly enter the system at this point.

*Period of Incubation.*—The period of incubation is shorter than that of any other infectious disease except perhaps influenza and diphtheria. The extremes range from a few hours to fifteen days. In 87 per cent. of cases Holt found the period to be less than six days, and in 66 per cent. between two and three days. In my own experience the incubation period has been short. In one case a child who had not been exposed either directly or indirectly came in contact with the disease in the late afternoon and developed the initial symptoms the following morning. Many of the cases of prolonged incubation present elements of uncertainty. The cases in which the incubation is longer than one week are extremely rare.

bloodvessels are collections of leukocytes. The production of epithelium is greatly increased during the acute stages, which results later in profuse exfoliation of the superficial layers. In the later stages, in addition to this, according to Neumann, there is also a profuse development of exudative cells, particularly among the ducts and follicles. These cells easily reach the epithelial surface, a fact which accounts for the great infectiousness of the desquamating cells. The throat changes in uncomplicated scarlet fever are catarrhal in nature, and are an essential part of the disease. The croupous and diphtheritic membranes must be considered as complications. The changes in the kidneys are those of a diffuse nephritis.

**Clinical Types.**—Scarlet fever is the most irregular of all the eruptive fevers in its severity and manifestations in different individuals. From the attack so mild that diagnosis is difficult to the fiercely malignant form we see every possible degree of severity. The majority of cases, however, pursue a fairly uniform course and may, with propriety, be called ordinary cases. Other types may be described as mild, severe, and malignant.

**Ordinary Type.**—In the ordinary or common type the onset is sudden and is characterized by vomiting, fever, sore throat, and rapid pulse. Occasionally a short period of malaise precedes the onset of definite symptoms. In older children a chill is sometimes the first symptom; in younger children a convulsion. The vomiting is usually repeated several times and may not be accompanied by nausea. When it occurs late in the disease it is a far more unfavorable symptom than at the outset. The intensity of the period of invasion is usually indicative of the severity of the attack, though this is a rule subject to many exceptions. The tongue is at first coated white. After three or four days it rapidly clears and becomes clean and red, with prominent papillæ, the true strawberry tongue.

Within twenty-four hours after the invasion and usually within twelve hours the characteristic eruption begins to appear. There is frequently intense itching or burning of the skin. The rash is usually well developed during the second day of its appearance. It then continues from four to six days, when it gradually subsides. It usually appears first over the front of the neck and upper part of the chest. It consists of minute points of bright-scarlet color closely grouped together on reddened skin. The points become confluent in places, forming bright colored patches, but over the most of the surface they remain discrete throughout. Being hyperemic in nature, the rash disappears on pressure leaving for a perceptible time a white spot.

Desquamation is perhaps the most characteristic symptom of all forms of scarlet fever. In no other disease does such extensive desquamation occur. Although in mild cases it is sometimes comparatively slight, always present if sought for. It rarely begins before the sixth day, is frequently delayed until the second week. It appears first on the hands and body or between the fingers. It begins as fine, branny scales, soon changes to large lamellar scales. Sometimes the skin is peeled off in strips. It continues from ten to thirty days, and is



PLATE XIV.



Eruption of Scarlet Fever.



persistent where the skin is thickest. It usually continues on the fingers and around the nails after other portions of the body are clear, which explains the readiness with which the disease is conveyed by letters.

*Mild Type.*—One of the most peculiar features of scarlet fever is its ability sometimes to appear in extremely mild form. The symptoms are sometimes so slight that medical aid is not sought, and in other cases diagnosis is difficult before the stage of desquamation. As a rule, however, there is an onset of vomiting, fever, and sore throat as in the ordinary type, but none of the symptoms is so urgent. The vomiting is not persistent, the temperature does not rise above 102° or 103° F., and the throat presents only the symptoms of mild pharyngitis. I have seen an undoubted case in which the temperature never rose to 101° F. The temperature may become normal on the fourth day. The eruption is often faint and may not appear on the face. It may, however, be bright and distinctive for twenty-four hours and then fade away so rapidly as to have disappeared by the fifth day. In rare instances it is an evanescent rash, which disappears entirely within twenty-four hours. Nephritis may be a sequel, due in many cases to exposure and lack of care, the natural results of so mild an illness. Owing to this lack of care and isolation, the patient may become very dangerous to others. It is by these mild cases that the disease is sometimes sown broadcast. A mild attack in one child may produce a malignant one in another. The "scarlatina" of the laity is often the cause of the disease in schools and institutions (Figs. 105 and 106).

*Severe Type.* This type differs from the usual form not only from the fact that the symptoms are aggravated, but the various stages are usually prolonged. The fever may continue for three weeks or more and the stage of desquamation for even a longer time. A fatal termination is common, death occurring usually during the second week. The chief peculiarity which distinguishes this from the ordinary form is the presence of septic symptoms due to streptococcic infection. The type might, therefore, with propriety be called the complicated type. The throat is usually first to show the evidence of streptococcic invasion. On the third day, and in some cases on the first or second day, a membranous exudate appears on the tonsils and soon invades the pharynx and nasopharynx. A purulent nasal discharge appears and the lymph nodes at the angle of the jaw begin to swell, the cellular tissues being so involved as to often cause immense enlargement. The Eustachian tubes are involved and purulent otitis media follows, but the larynx commonly escapes. The urine contains albumin, perhaps blood cells and hyaline and epithelial casts. Symptoms of general septic infection rapidly supervene. There is low delirium or stupor; the child refuses nourishment and may die from exhaustion, but sudden death is not uncommon. Others, after overcoming one symptom after another, slowly recover after a tedious convalescence.

This type often differs so radically in its symptoms from the uncomplicated type as to seem like another disease. In the one we have an infectious disease, running a definite course and presenting few urgent

symptoms. In the other we have a typical picture of septic infection. The countenance is gray or of the greenish-yellow, septic hue. The breath is fetid and there is an offensive discharge from the mouth and nose. The fever is high, the pulse rapid and weak, and there is either stupor or delirium. Sordes collect upon the teeth, the mouth is sore and the head is thrown back to relieve the dyspnea. Albumin appears in the urine and cardiac or pulmonary complications are apt to supervene. The appearance of the patient is usually quite different from that of the one passing through an uncomplicated attack.

The disease occasionally appears in severe but not strictly malignant form in which there are no complications, but the patient is placed in great danger or dies from the severity of the disease itself. In some epidemics such cases are comparatively common. In still other cases the local symptoms are severe, but the general septic infection is mild.

*Malignant Type.*—This form of the disease is fortunately rare. It was formerly without doubt more common than it is now. Hence, scarlet fever was a more dreaded disease forty or fifty years ago than it has been during the past twenty years. This, at least, seems to be true in the Eastern United States. The scarlatinal poison may be so intense as to cause death within twenty-four hours. More commonly death does not occur before the third or fourth day, the patient being comatose or delirious. The nervous symptoms are so marked that some physicians have given this form the name of cerebral scarlet fever. In a case of my own the initial symptoms were convulsions, hyperpyrexia, and hematuria. In another case hyperpyrexia and coma appeared at the outset, the patient dying soon after the rash began to appear. In an epidemic occurring in the practice of my father about forty years ago, the eruption was hemorrhagic in character and the patients died within the first two or three days. The peculiar eruption and cerebral symptoms led some physicians in the early stages of the epidemic to make a diagnosis of cerebrospinal meningitis. Although a temperature of 106° or 107° F. is commonly seen in such cases, a very low temperature sometimes occurs, with great prostration. The scarlatinal poisoning is so intense in these cases that the patient seems to be overwhelmed by it. Death results from the intense poison of the disease rather than from complications.

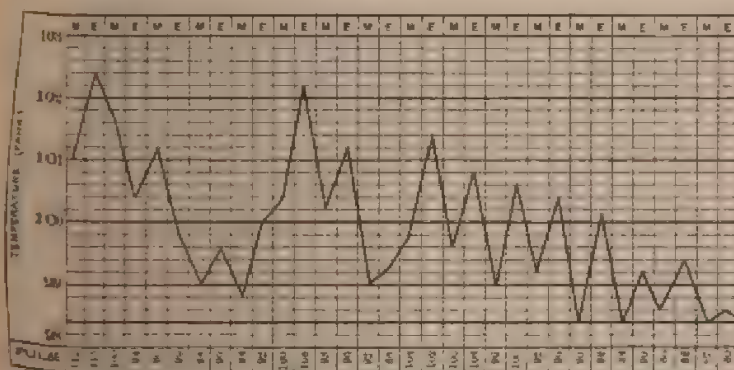
*Surgical Scarlet Fever.*—Patients who have undergone surgical operations are unquestionably very susceptible to scarlet fever. Such scarlet fever, however, is not essentially different from the ordinary disease. It is simple scarlet fever in a surgical case. It is no doubt true, as Osler has shown, that the eruption which has frequently led to a diagnosis of scarlet fever is nothing more than the red rash of septicemia. It is a fact that surgical scarlet fever is less common since surgical septicemia has become less frequent. Hoffa has attempted to make a classification of the rashes which are seen in surgical cases. These he divides into three classes as follows: (1) Those due to vasomotor irritation and seen chiefly after operations where the nerve supply is abundant. The rash occurs within a few hours and resembles an



erythema and usually disappears after a few hours. (2) "Toxic erythema," which appear usually on the second day after operation without prodromal symptoms. There is frequently fever and gastric disturbance. The rash may be simply a diffuse redness or there may be large, isolated red patches. It frequently disappears within twenty-four hours without desquamation. This condition is due to the absorption of wound secretions like fibrin ferment and is analogous to the eruption following the administration of such drugs as antipyrin or copaiba. (3) The eruptions of pyemia and septicemia which indicate general septic infection. They may be diffused or in patches and sometimes closely simulate the eruption of scarlet fever.

True surgical scarlet fever is usually atypical in its manifestations. The throat symptoms are not always characteristic and the rash is often irregular in its appearance and manifestations. The constitutional symptoms are frequently grave in nature. While caution should

FIG. 165



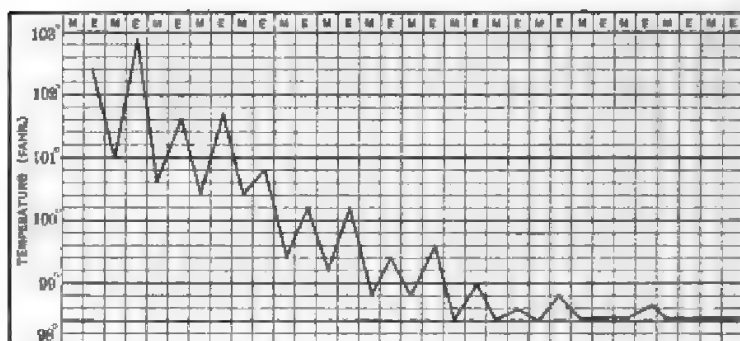
Very mild scarlet fever in a boy of five years, complicated by otitis with discharge from the left ear on the sixth day and from the right ear on the ninth day.

be exercised in allowing a case to pass unrecognized, the diagnosis should certainly not be made in atypical cases in which no desquamation follows.

**Symptomatology. Invasion.**—The invasion of scarlet fever is usually characteristic, but is subject to many variations. In typical cases the invasion is usually more abrupt than in most diseases. The vomiting, sore throat, high temperature, and rapid pulse are a combination of symptoms which should always put the physician on his guard. Either of these symptoms, however, may be absent. In my last three cases there was no vomiting at any time and in two of them the sore throat was not marked. Scarlet fever is often mistaken, at the first visit for tonsillitis. The sudden fever, malaise, and sore throat, in conjunction with tonsils covered with a punctate exudation, make some cases appear like tonsillitis. In a considerable number of cases the onset is gradual and

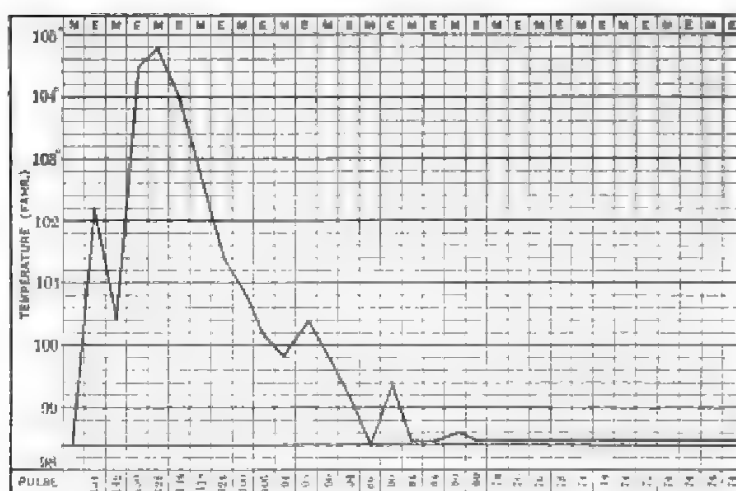
the early symptoms are indefinite. In general terms the more severe the attack the more distinctive the onset. In rare cases a chill is the

FIG. 106



Uncomplicated scarlet fever of mild type in a girl of six years.

FIG. 107



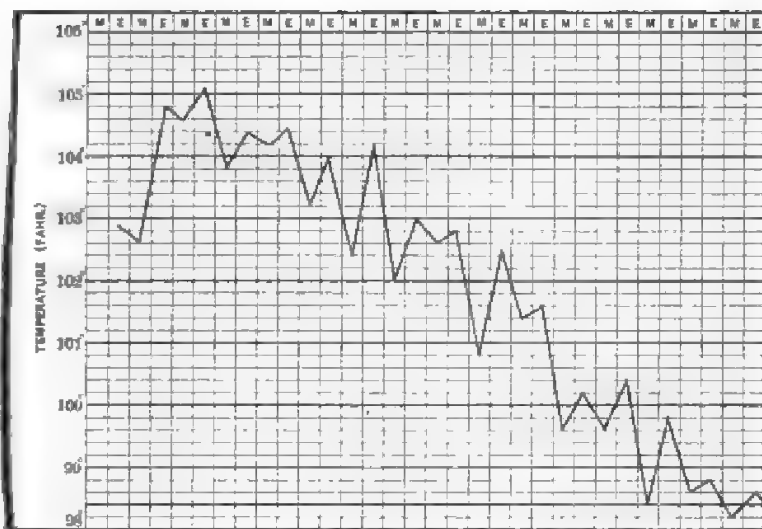
Uncomplicated scarlet fever of moderate severity in a boy of twelve years, marked by a prodromal stage of lassitude and nausea lasting six hours before the advent of vomiting and fever; follicular spots on each tonsil with moderate rash on the second day; follicular spots almost invisible, nausea ceased, but rash intense on the fourth day; desquamation on the body on the eighth day, becoming profuse two days later; desquamation beginning on the fingers and toes on the eleventh day; desquamation complete on the body, but still free on the hands and feet on the twenty-fourth day; released from isolation on the thirty-sixth day.

first symptom. This is more common in older children and adults. In young children a convulsion may be the initial symptom.

*Temperature.*—There is no typical temperature range in scarlet fever as there is in such diseases as pneumonia and typhoid fever (Figs. 106

to 110). It is a disease, however, in which the temperature usually ranges high. The height of the temperature at the onset is to a certain extent an indication of the severity of the attack. A temperature on the first day above  $104.5^{\circ}$  F. gives promise of a severe attack; below  $102^{\circ}$  F., of a mild attack. The temperature usually reaches its highest point on the second or third day in uncomplicated cases. It is frequently remittent and in mild cases almost intermittent. Occasionally the highest temperature will be found on the first day. A falling temperature after the first or second day is indicative of a mild attack. A rising temperature after the third or fourth day usually indicates a compli-

FIG. 108



**Uncomplicated scarlet fever** in a girl of seven years, running a somewhat prolonged course of more than usual severity; onset sudden with rash distinctly marked within twelve hours, reaching its height on the third day, continuing for four days and disappearing only at the end of the tenth day; desquamation beginning on the eleventh day and lasting on the hands and feet until the forty-sixth day; a grayish-white exudate on the tonsils on the third day, increasing on the following day, and then slowly disappearing; moderate adenitis from the fourth to the eighth day.

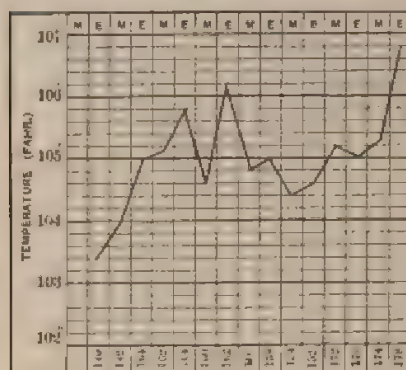
cation and is always a warning signal which should not be neglected. Normally the temperature, even in severe cases, begins to subside as the rash begins to fade. Any departure from this general principle is usually indicative of a complication.

**Pulse.**—A rapid pulse is characteristic of scarlet fever. I have come to look upon it as an aid in making a diagnosis in uncertain cases. A pulse of 120 with mild and perhaps not urgent symptoms is not uncommon. It is frequently found to be 140 or 150 at the first visit, and while by no means pathognomonic, it is certainly most suggestive.

**Throat.**—Sore throat is one of the most constant of the initial symptoms of scarlet fever. As already stated it is frequently mistaken for

tonsillitis at the outset. In such cases an exudate appears early and is liable to be in patches. In such throats there is almost invariably more

FIG. 109



Scarlet fever in a girl of three years complicated on the third day by extensive membranous angina with extreme adenitis and cellulitis and by pneumonia on the seventh day; death on the eighth day.

almost invariably truly diphtheritic in nature. Diphtheria sometimes appears at the outset also. A diagnosis cannot always be made without a bacterial culture. Streptococci, staphylococci, and Klebs-Loeffler bacilli may all be present. The angina when severe is usually accompanied by a discharge from the nose of a clear, tenacious mucus or mucopus. This may go on to cause complete obstruction of the nasal passages. Such a condition is very prone to be followed by otitis. The more decidedly purulent the nasopharyngeal inflammation, the greater the danger of otitis. The simple angina of scarlet fever as well as the nasopharyngeal symptoms reach their height coincident with the eruption and the other symptoms, and gradually subside as improvement occurs in other directions. In many cases there is no exudate in the throat during the whole course of the disease. There is diffuse redness with fine, dark macules, but nothing more. There may even be considerable swelling without the appearance of any membrane. In some cases the child complains of severe soreness when there is nothing to be seen except diffuse redness.

Membranous sore throat is not a necessary part of scarlet fever. Many cases pass through their entire course without showing any membrane. It must, therefore, be considered a complication and not an essential feature of the disease and will be considered in a later section.

*Adenitis.*—In most cases of scarlet fever of ordinary severity the lymph nodes at the angle of the jaw are somewhat involved. This may occur even in the milder cases. They may be felt as small kernels and are frequently not sore. When the angina is severe, however, they become more seriously involved and may go on to acute inflammation

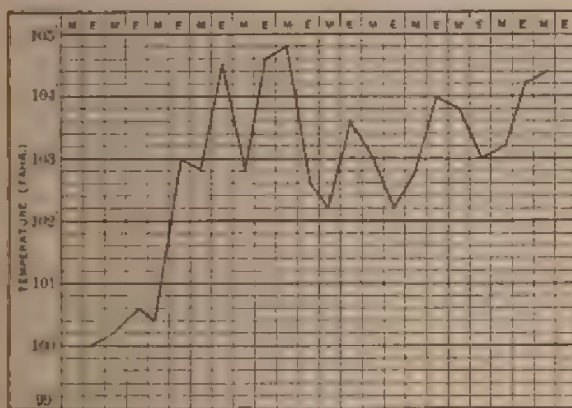
swelling and diffuse redness than is seen in tonsillitis. The congestion extends forward onto the hard palate and the uvula is swollen and is sometimes edematous. In other words, the inflammation of the throat is usually more extensive and severe than it commonly is in tonsillitis. As a rule, the exudates do not appear until the third or fourth day. They are then less like the exudates of tonsillitis, being smeared over the tonsils and adjoining tissues in irregular patches. The exudates which appear during the first week are usually caused by streptococci or staphylococci, and are, therefore, pseudodiphtheria. The exudates which appear late in the disease are



or suppuration. When this occurs there is usually marked cellulitis as well. While slight involvement of the lymph nodes is the rule, the more serious disorders are not essential to the disease and are to be considered as complications.

*Eruption.*—The eruption of scarlet fever presents many peculiarities. It is simulated by many other eruptions and is sometimes so atypical as to give but little aid in making a diagnosis. It is frequently apparent on the neck and chest within twelve hours of the initial symptoms and it is commonly present on the morning following the day upon which the illness began. In rare cases it is delayed for more than thirty-six hours, but very rarely does it appear after the fourth or fifth day. Among 108 hospital cases Holt found that the rash continued from three to seven days in

FIG. 110



Severe septic scarlet fever in a girl of nineteen months, beginning mildly but being complicated second day by an extensive putrid membranous angina and rhinitis and signs of septic infection, and ending by pneumonia and death on the twelfth day. Staphylococci and pneumococci were found in large numbers in the throat cultures on the fourth day and streptococci on the sixth day, but no *Klebsiella* bacilli were present at any time. The cervical cellulitis was extensive; there was extreme opisthotonus with rigidity of the legs and arms, and from the seventh to the tenth days there were several general convulsions.

81 The rash covers the face and whole body and has usually reached its height at the end of twenty-four hours. A peculiar pallor about the mouth is a characteristic feature of the disease.

An eruption of fine vesicles is seen in rare instances and occasionally a blotchy eruption appears on the face, but subsides as the typical rash develops. The intense itching which frequently is present when the rash is developing, particularly if accompanied by fine vesicles, sometimes renders the appearance of the case quite different from that of the regular form. The rash is sometimes very faint. In some mild cases where the disease is not suspected and medical aid is not sought in the early stages no history of a rash can be obtained. In such cases the rash is usually most marked in the axillæ and groins or other folds of the skin. At times it is irregular in its distribution, appearing in large,

bright patches in some regions and being very faint in others. In some cases as it does not appear on the face it is overlooked unless sought for on the body. When the rash is faint or uncertain, a hot bath or the application of hot water to a part of the body may cause it to show. The common fear of the laity that the rash will not come out well or that the condition is unfavorable when the rash is faint is not well founded. It is true that a rash that suddenly subsides or becomes faint is indicative of heart failure, but when the symptoms are mild, a faint rash need cause no anxiety.

On the other extreme from these mild cases, in which the rash is faint or the irregular ones in which it does not appear in typical form,

FIG. 111



Well-marked desquamation upon the dorsum of hands and fingers. (Welch and Schamberg.)

are the malignant cases in which the rash does not appear or appears in hemorrhagic form. In malignant cases the child sometimes dies before the rash appears; in others the rash is atypical in appearance actually hemorrhagic. These latter conditions, however, are very rare. There may be considerable edema or swelling of the hands and feet when the eruption is intense. It is not, however, a symptom of particular gravity.

*Desquamation.*—This is the most positive sign of scarlet fever. the peculiar extensive desquamation occurs in no other condition (Fig. 111). It rarely begins before the sixth or seventh day and sometimes much later than this in its appearance. There is frequently therefore, an interval between the disappearance of the rash and the appearance of the desquamation when the skin seems normal. A

PLATE XV.



Scarlet Fever Desquamation. Sixth day of the disease.  
(Welch and Schamberg.)





ient seen for the first time during this interval may mislead a physician. If the case has been mild and the early symptoms have been obscure, when the skin has received careful attention and is oiled daily the desquamation may be almost imperceptible and no definite scales appear, they form with the oil little rolls or balls as the skin is rubbed. With the best care there is but little dissemination of the infection. Desquamation of the trunk and extremities is not infrequently complete in a week, and there is sometimes an interval of several days before desquamation of the feet and toes begins. Desquamation of the fingers usually begins a little earlier than it does upon the feet, partly perhaps because

FIG. 112



Epidermal glove-like casts from a fatal case of scarlet fever. (Welch and Schamberg.)

A child has a tendency to pick continuously at them. It begins first at the ends of the fingers (Fig. 111). The finger-tips are frequently red, soft and pink, while the rest of the hand is covered with a grayish, thick skin, with white patches where the skin is loosening. As a rule, the skin is picked off by the patient in little patches and shreds, but cases are on record in which quite complete casts of the fingers and even of the hand have been thrown off (Fig. 112). This desquamation in reds and strips is rarely ever seen on the unexposed portions of the body. Desquamation of the hands and feet is very rarely complete before the thirtieth day after the onset of the disease. It more frequently

requires forty, and sometimes persists about the nails until the end of seven or eight weeks, as mentioned under chart (Fig. 108). Extensive desquamation of this character, and particularly the lamellar desquamation of the hands and feet, is so characteristic of scarlet fever that a diagnosis may be made from it even when the early symptoms are obscure and uncertain. Even if the desquamation is somewhat uncertain, no child showing it should be allowed to mingle with others.

In rare cases a second desquamation occurs and even a third, which, of course, prolongs the course of the disease. These secondary desquamations are usually not as extensive as the first. They commonly involve a portion of the body, but a second general desquamation of the body has been known to occur.

*The Urine.*—In the Paris Theses of 1903, Lahdé reports observations made upon the urine in scarlet fever and diphtheria. In scarlet fever diminished secretion continues during the first six days of the eruption, but there is a sudden increase at the eighth or ninth day. The acidity is decidedly increased. The minimum excretion of urea occurs at the fifth day of the eruption. There is then usually a sudden increase followed by a gradual fall. The presence of urobilin and indican is rare in scarlet fever, but is constant in diphtheria. The diazo reaction is occasionally found in scarlet fever, but is never seen in diphtheria. During the febrile stage a slight amount of albumin is found frequently in the urine and sometimes blood and hyaline casts. This is most commonly febrile albuminuria, which usually disappears as the fever subsides. Except in the more severe forms, suppression is rare and dropsy more so. These symptoms usually subside as the fever falls. The kidney symptoms at this stage rarely prove serious. They may, however, do so and always demand attention. The more serious kidney symptoms occur later and will be considered as complications.

*The Tongue.*—The term "strawberry tongue" has misled many young practitioners. One author describes it as a white-coated tongue, showing prominent red papillae; while another says that it is a rough, red tongue, presenting dark-red papillae. These two conditions are very different, so different in fact that they cannot be diagnostic of the same pathological condition. Fussell has recently quoted the description of the strawberry tongue given by over twenty authorities, which show the greatest divergence of opinion. The consensus of opinion would seem to be that the term should be applied, not to the white-coated tongue, frequently seen in the first stage of scarlet fever, but to the red, rough tongue commonly seen on or after the fourth day, the papillae being dark red in color. There are two facts worthy of consideration: A white tongue with red papillae is seen in numerous conditions and is not confined to scarlet fever; the strawberry is not a white fruit with red seeds, but a dark-red fruit with a rough surface. It is unwise, therefore, to apply the term strawberry to a white tongue, and still more unwise to lay stress on such a tongue as a symptom of importance in making diagnosis of scarlet fever.

The true strawberry tongue was originally described by Flint

follows: "The tongue early in the disease generally is coated. While the coating remains, frequently the papillæ projecting through it have the appearance of red points; the surface of the tongue looks as if cayenne pepper or red sand had been sprinkled over it. This is seen in other affections. Another appearance is quite distinctive of scarlet fever. In the progress of the disease the coating exfoliates, leaving the surface of the tongue clean and reddened; and the papillæ being enlarged, the appearance is strikingly like that of a red strawberry. The strawberry-red tongue is a pathognomonic symptom; it is peculiar to the disease. It is often but not uniformly present."

**Constitutional Symptoms.**—The constitutional symptoms of scarlet fever are in no way characteristic. They vary with the severity of the attack and with the presence or absence of complications. There is occasionally an indefinite premonitory stage in which the child seems ill, but shows no characteristic symptoms. In a recent case of my own (see Fig. 107) there was so marked a change in the color and appearance of the child that the mother took the temperature at noon and found it normal. At five o'clock there was vomiting and the temperature was 102.2° F. The rash was present on the following morning. Headache is frequently present, but is not constant. There may be aching of the limbs and muscular pains, but these are also not constant symptoms.

**Complications and Sequelæ.**—The common complications of scarlet fever are angina, otitis, adenitis, arthritis, and nephritis. The most common sequelæ are nephritis and deafness.

**Angina.**—Except in the mildest cases the throat always shows more or less pathological change. A catarrhal condition of the throat is normal to scarlet fever, but membranous exudates and gangrene are not essential to it. Small white or yellowish spots similar to those seen in tonsillitis sometimes appear before the eruption and may readily lead to a diagnosis of simple tonsillitis. These spots may coalesce to form membranes, or they may wholly disappear after two or three days. This simple angina cannot be considered a complication. Two other forms of angina, however, frequently occur and are, strictly speaking, complications. These are the membranous angina and gangrenous angina.

Bacteriologists have settled that with few exceptions the angina of the early stages is pseudodiphtheria, that of the late stages true diphtheria. While primary pseudodiphtheria is a mild disease, the death rate being rarely over 5 per cent., secondary pseudodiphtheria is very dangerous and fatal. The membrane may appear in the throat on the first or second day, but is not usually seen before the third day. It is generally confined to the tonsils, but frequently fills the throat and nasopharynx. It shows a tendency to invade the ears and nose and to shun the larynx. It reaches its height about the sixth or seventh day. It frequently presents all the local characteristics of diphtheria, together with the general symptoms of septicemia. The pseudomembranes vary considerably in color. They are commonly of a gray or greenish cast, but are occasion-

ally clear and white. In severe cases the exudate is sometimes black. Even when not strictly gangrenous, the breath may be of a foul and sickening odor and there is a thin, fetid discharge from the throat and nose. There is frequently edema in and about the nasopharynx which renders mouth breathing necessary. These changes in the throat are invariably accompanied by swelling of the lymph nodes and cellulitis. When the throat symptoms are marked the constitutional symptoms are also severe as a rule, owing to general septic infection in which streptococci play the most important part. When the throat symptoms are severe, otitis almost always occurs and pneumonia and nephritis are particularly common (Figs. 109 and 110).

Without a bacterial examination it is frequently impossible to distinguish between membranes due to the Klebs-Loeffler bacillus and those resulting from some other germ. As a rule, streptococcus anginas are accompanied by more inflammation, edema, and redness, and by greater infiltration of the tissues of the neck than in true diphtheritic angina. In the streptococcus disease there is a strong tendency to invade the ears, while in diphtheria the larynx is more commonly the direction of extension. The exciting cause of membranous inflammation is usually the streptococcus pyogenes. It is occasionally associated with the staphylococcus aureus or albus, but the streptococcus is the more commonly observed. It occurs not only in the pseudomembranes and the tissues under it, but is found in the blood in large numbers. Through the agency of the toxins which it generates it is unquestionably the cause of many of the complications and of general septicemia.

The pseudomembranes, which appear late in the disease, are usually associated with the Klebs-Loeffler bacillus. Diphtheria is in the fullest sense of the word a complication, and is not an essential symptom of scarlet fever.

Gangrenous angina is the worst phase of the scarlatinal sore throat. The symptoms already described are exaggerated. The odor of the breath is very offensive. The swelling and obstruction in the throat are extreme and the infiltration of the lymph nodes is very great. The throat is filled with a pultaceous mass of sloughing tissue, of gray or blackish color. The discharge from the throat and nose may be purulent or thin and fetid. The prostration and symptoms of general infection are extreme; the case usually terminates fatally. In extreme cases the slough sometimes involves a bloodvessel and sudden death may occur from hemorrhage.

*Otitis.*—The most common complication of scarlet fever next to angina is otitis. Its effects are often serious, for it is a common cause of deaf-mutism. It results from the extension of the inflammation from the throat through the Eustachian tubes. The tendency to ear involvement varies with different epidemics, but it is more common in young patients. It does not usually occur until the second week, and, as a rule, involves both ears. Its presence may be indicated by earache and an increase in the fever, but frequently a discharge is the first indication.



that the ears are involved. The process is prone to be a destructive one and to result in long-continued suppuration. It sometimes leads to a rapidly fatal meningitis. When it occurs before the fever has subsided it may produce no symptom and the child may complain of no pain. When it occurs as a later complication its advent is usually marked by fever and increased pulse rate. When the membrane does not rupture and the tension in the cavity of the middle ear is very great, nervous symptoms may become pronounced, muscular rigidity, opisthotonos, and even convulsions being possible. When destructive ear changes occur in early childhood, deaf-mutism is a probable result. Among 5613 deaf-mutes whose cases were investigated by May, it was found that 572 cases resulted from the otitis of scarlet fever.

*Adenitis and Cellulitis.*—Adenitis and cellulitis are not unusual results of streptococcic invasion of the throat. Not only are the lymph nodes themselves enlarged, but there is more or less inflammatory edema of the surrounding tissues. That this is due to secondary infection is shown by the fact that the streptococci are found in abundance in both the nodes and edematous tissue around them. Enlargement of the nodes may be detected during the first week, but serious cellulitis does not, as a rule, occur until later in the disease. Suppuration, sloughing, or even gangrene may occur. The cellulitis may be localized to a small area around the enlarged lymph nodes or may be general. As soon as it becomes marked the head is apt to be drawn backward. In extreme cases this is so conspicuous a symptom as to lead to a suspicion of cerebrospinal disease. Dyspnea is not uncommon. An extreme suppurating adenitis is a complication of the utmost gravity. It may lead to death by involving important vessels or by the slower process of general infection. As it usually accompanies an extreme and serious throat complication, the prognosis is always bad.

*Arthritis.*—Scarlatinal rheumatism has been relegated by modern methods of investigation to the list of rare diseases. The joint affections which occur during the course of scarlet fever are, however, not uncommon, but they are proved to be due to more than one pathological condition. True rheumatic arthritis is one of the rarest of the joint affections complicating scarlet fever. The most common joint lesion of scarlet fever is synovitis, and the next most common is probably septic arthritis. In classifying these various lesions, Marsden, of London, uses Ashby's classification, slightly changing the nomenclature as follows: (1) scarlatinal synovitis; (2) septic arthritis; (3) acute or subacute rheumatic synovitis; (4) tuberculous arthritis.

Scarlatinal synovitis usually appears early in the second week, continues for three or four days, and disappears. Suppuration is rare; it is seldom seen under four years. The onset is usually acute and, as a rule, the attack runs an acute course. In some cases there is nothing to be found save pain on movement, or tenderness; in others the whole of the hand is red and swollen. Between these two conditions all grades are met. The hands and wrists are the favorite site. They were attacked in no less than 72 out of one series of 100 cases. Any joint, however,

may be affected. Septic arthritis is frequently known as pyemic arthritis. In this condition the large joints are usually involved and the lesions are apt to be multiple. The condition is always a grave one, as suppuration and injury to the joint are common. True rheumatism occurs third in order of frequency. It appears late in the course of the disease and rarely proves serious. The attack is almost invariably subacute in character and continues for a few days. Evidence of heart involvement is not uncommon and a permanent murmur is sometimes left behind. Antirheumatic treatment usually gives prompt relief.

*Nephritis.*—During the acute stage, particularly when the fever is high, a slight amount of albumin is usually found. It is commonly only a slight febrile albuminuria due to degenerative nephritis which subsides as the temperature falls. In the grave type kidney lesions may occur, to which the term septic nephritis has been given. The urine contains albumin, but blood and casts are not necessarily present, neither do the rational symptoms of uremia appear.

The most characteristic and common kidney lesion is postscarlatinal nephritis, which is a diffuse nephritis. It usually develops during the third or fourth week. There may be no interval of apyrexia between the kidney attack and the nephritis. It may be so mild as to almost escape notice, or it may be so severe as to cause death. Recovery may be complete or incomplete. The first symptom to be noticed is usually edema of the face, which is frequently accompanied by feverishness and restlessness. Dropsy and all the characteristic symptoms of acute nephritis rapidly develop. The urine usually shows a small amount of albumin for a few days before the advent of definite symptoms. As the disease develops the urine becomes scanty and high colored and may be completely suppressed. It contains a large amount of albumin and is loaded with blood cells and casts. The first evidence of albumin after the second week of scarlet fever should be a warning of danger, and should receive immediate attention.

Daily examination of the urine is desirable. It is a wise plan to take to the house of the patient test tubes, a spirit lamp, and a bottle of nitric acid. A pocket test case is useful in these cases. An examination may thus be made daily with but little loss of time, as the early detection of albumin always repays for the trouble taken.

*Other Complications.* Numerous other pathological conditions may occur as complications or sequelæ, but are less common than those mentioned. *Pneumonia*, although commonly found at the autopsy of patients who have died with septic symptoms, is frequently not recognized before death. *Pleuropneumonia* occasionally occurs when there is marked septic infection. Either simple or pleuropneumonia is a grave complication and usually determines a fatal result. *Empyema* is also a possibility in septic cases or as a sequel of pleuropneumonia. When there is marked nephritis, serous effusion into the pleural cavity may occur, and edema of the lungs is not uncommon as a terminal symptom. *Endocarditis* and *pericarditis*, though uncommon, are sometimes encountered. Murmurs are occasionally heard during the course

of the disease, which disappear as the active symptoms subside. Permanent organic lesions sometimes develop in conjunction with the late kidney complications. The various serous membranes are occasionally involved. Endocarditis is rather more prone to be of the malignant type in scarlet fever than it is in simple rheumatic cases. As in all diseases marked by high temperature or septic infection, myocarditis is not uncommon, and acute dilatation of the heart is sometimes encountered.

*Nervous symptoms* are less frequent than might be expected in a disease so often septic in its nature. A convulsion in rare cases occurs as an initial symptom. Convulsions due to uremia sometimes occur in the late stages. In a recent case under my observation, the child for thirty-six hours showed marked opisthotonos. There was contracture of the muscles of the extremities, with repeated convulsive attacks of the nature of tetany. These convulsions, some of them severe, were precipitated by anything which irritated the child—such as attempts at feeding or syringing the nose. Meningitis is rare, but retraction of the head due to swollen lymph nodes sometimes leads to the belief that it is developing. It may occur as the sequel of otitis and even as a complication of nephritis. Chorea is very rare even when the case is complicated by diphtheria. Peripheral paralysis is also rare.

*Vomiting* usually occurs at the outset and the stomach is sometimes irritable for two or three days, but grave gastroenteric disturbance is not common, except in malignant cases. Loss of appetite during the period of fever is not uncommon, and feeding is a difficult problem. Catarrhal *stomatitis* is of frequent occurrence, and this, together with sore throat, frequently leads the child to refuse food when it might otherwise accept it.

Except in very mild cases leukocytosis is present. Even in such cases it may occur to a slight degree. Whenever suppuration occurs the leukocytosis increases. A marked leukocytosis, therefore, is to be expected in complicated cases. The blood conditions in this disease are fully described in the Section on the Blood. *Purpura hemorrhagica* and peculiar attacks of superficial symmetrical *gangrene* have been reported in a very few cases. The thighs and arms are most commonly affected in this latter disease, which runs a rapid and usually a fatal course.

*Other Exanthemata as Complications.*—Scarlet fever may be complicated by any of the other infectious diseases. After diphtheria, measles is probably the most frequent of these, but chickenpox, smallpox, typhoid fever, and erysipelas have been reported as occurring coincidentally with scarlet fever. When two of these diseases occur synchronously, the symptoms are obscure and often puzzling. As a rule, however, the onset of one disease occurs as the other is beginning to subside, and the two eruptions succeed each other. The tendency of diphtheria to complicate scarlet fever has already been dwelt upon. It usually occurs after the scarlet fever has partially run its course, but I have seen it precede the scarlet fever.

**Diagnosis.**—In typical cases the diagnosis of scarlet fever is very easy. It is the irregular forms which cause uncertainty. In all the exanthem-



ata it is usually peculiarities in the eruption which render the diagnosis most difficult. In doubtful cases it is impossible to make a diagnosis from the eruption alone. There are many simple rashes due to digestive disturbance or mild infection which closely simulate scarlet fever. It is occasionally necessary to wait for the period of desquamation before a positive diagnosis can be made. As a rule, too much attention is devoted to the eruption to the exclusion of other symptoms. The eruption produced in some cases by belladonna or atropine, quinine, antipyrin, and occasionally by diphtheria antitoxin is much like that of scarlet fever. Due consideration of the accompanying symptoms, however, is usually sufficient to prevent error in diagnosis. Certain types of urticaria and simple eruptions of that class are also occasionally very suspicious. If accompanied by digestive disorders with vomiting and fever, the diagnosis is sometimes very difficult. An erythema occasionally accompanies typhoid fever, which may lead to an erroneous diagnosis. There have been certain epidemics of influenza in which a scarlatiniform erythema has caused much anxiety to the medical attendant. The sudden onset of fever, with sore throat and perhaps nausea followed by a more or less extensive erythema is a picture very suggestive of scarlet fever. In my experience the eruption in these cases has been coarser than that of scarlet fever and there has been less diffuse redness. Occasionally, however, a uniform erythema without the red pinhead points has been present. "Grippe with a rash" presents some very difficult cases for diagnosis. The pulse in scarlet fever is more rapid than in influenza.

Occasionally the rash of scarlet fever is in places blotchy. Usually, however, if search is made areas of reddened skin will be found dotted with the characteristic pinhead spots. These areas are more apt to be found in the groins and axillæ and in folds of the skin. When the rash is faint, a hot bath may sometimes render the diagnosis easy. The same result may be accomplished locally by placing cloths wrung out of hot water for a few minutes across the abdomen or chest. When the temperature is very high in some cases of the malignant type the rash is hemorrhagic. This, together with the nervous symptoms, may lead to the suspicion of epidemic cerebrospinal meningitis. A white line appearing at the junction of the finger-nail and the pulp of the finger is considered by McCollom a valuable sign of scarlet fever. Desquamation is undoubtedly the most distinctive feature of scarlet fever, but it is unfortunately a very late one. A rash, if it is ever so mild, if followed by characteristic desquamation of the hands and feet, may be considered as certainly scarlatinial. If no desquamation appears after careful watching, it is almost equally certain that the case was not scarlet fever. Attention is called on another page to the fact that scarlet fever is sometimes mistaken during the first twelve or twenty-four hours for tonsillitis. The early throat symptoms of scarlet fever are often very similar to those of diphtheria. In many cases it is impossible to make a diagnosis without a bacterial culture. The presence of diphtheria at the outset is entirely possible, but in the large proportion



of cases the exudates of the early stage are pseudodiphtheria. In scarlet fever urobilin and indican are rarely found in the urine, but are constantly found in diphtheria. In scarlet fever the diazo reaction can frequently be obtained, but never in diphtheria.

The diagnosis between scarlet fever and measles rarely offers any difficulties. The prolonged prodromal stage of measles, with its coryza, cough, and suffusion of the eyes, followed by a blotchy, slow-spreading eruption, forms a picture so characteristic that it is rarely mistaken for scarlet fever. This is not as true, however, regarding rubella. Some cases of this disease are very difficult to distinguish from scarlet fever. On the other hand, mild scarlet fever is not infrequently mistaken for German measles. In rubella there are usually no prodromal symptoms. Vomiting, sore throat, fever, and rapid pulse are all wanting. The eruption is the first symptom to appear. It appears first on the face and looks much like that of scarlet fever, but is usually less markedly punctiform. It is more diffuse and a little lighter in color. If the whole body is examined areas will usually be found in which the eruption is coarser and loses its scarlatiniform aspect. Desquamation is absent or appears in very fine, branny scales. Enlargement of the cervical and auricular lymph nodes is almost invariably present in rubella, but is rare in scarlet fever. The most important point in differential diagnosis is the absence in rubella of constitutional symptoms. Although very mild cases of scarlet fever are sometimes seen, a rash, as bright and distinct as that of the average case of rubella, is invariably accompanied for a day or two at least by distinctive constitutional symptoms. The pulse is rapid and the temperature rarely below 102° F.

**Recurrence and Relapse.** While second attacks of scarlet fever sometimes occur they are extremely rare, probably more so than in the case of any other infectious disease. So many other rashes simulate that of scarlet fever that errors in diagnosis are not difficult. The reports of second attacks must be received with much reservation, and are to be unreservedly accepted only from competent and cautious observers.

Relapses are more common than second attacks. They result from autoinfection and occur during the second or third week. They are similar in their nature to the relapses of typhoid fever. They sometimes pursue the course of the primary disease. As in the relapses of typhoid fever, they are frequently less severe than the primary attack, but this is not always the case. These true relapses should not be mistaken for those cases in which the rash subsides for a few days and then reappears. This latter condition sometimes occurs with the increased fever which accompanies a late complication. I have seen a rash which had almost disappeared reappear very distinctly upon the administration of a hot bath and continue clear for more than twenty-four hours.

**Prognosis.**—After a study of a large number of American and European cases, Holt concludes that the general mortality of scarlet fever may be assumed to be from 12 per cent. to 14 per cent., while under five years it is from 20 per cent. to 30 per cent. It is much lower in private practice than in hospitals, and varies greatly in different epidemics. Statistics

as to general mortality rates give but little practical aid in determining the prognosis of any particular case. The two most important general factors are the age of the child and the character of the prevailing epidemic. The younger the patient, the greater the danger. The majority of fatal cases occur in children under seven years. In a study of 1000 cases, J. H. McCollom found the mortality of all cases to be 9.8 per cent. Scarlet fever unaccompanied caused 58 deaths; bronchopneumonia, 15; diphtheria and scarlet fever combined, 10; diphtheria alone, 9; pneumonia, 4; scarlet fever and erysipelas, 1; tuberculous meningitis, 1.

Death may occur at any stage from the outset until months after the subsidence of acute symptoms. Death during the first few days usually occurs only in the malignant cases in which the patient is overwhelmed by the poison of the disease itself. Death in these cases is due strictly to scarlet fever. Death during the second and third weeks may also result from the intensity of the scarlatinal poison, but is more commonly due to some complication, especially diphtheria, pneumonia, and acute nephritis. It may result also from intense septic infection due to severe throat and glandular involvement, death being due to exhaustion. Death after the third week is usually due to postscarlatinal nephritis. This may occur without reference to the severity of the early stages and may be postponed for weeks or months after the disease has run its course. Prognosis is rendered unfavorable by the appearance of the following symptoms, the gravity being in proportion to their severity: violent onset, high temperature, convulsions, extensive pseudomembranes, gangrenous pharyngitis, diphtheria, croup, pneumonia, excessive cellulitis, superficial gangrene, nephritis, and exhaustion, with general septic symptoms. The prognosis in uncomplicated cases in older children, even when the disease runs an active course, is good.

**Prophylaxis.**—In few other diseases are preventive measures so productive of good results as in scarlet fever. Its spread can be more readily controlled than can that of most of the other infectious diseases. The measures necessary, however, to that end are many in number and very complex, and demand on the part of the practitioner much thought and perseverance. When we consider the high mortality of scarlet fever and the grave sequelæ in those who survive, we are forced to feel that neglect of preventive measures is little short of criminal.

Every child who is known to have been exposed to scarlet fever should be isolated. It is true that the disease is not contagious during the period of incubation. It is doubtful, indeed, whether it is contagious before the appearance of the eruption. Children in contact with scarlet fever patients for several hours after the initial vomiting do not always contract the disease. There may be exceptions, however, to the rule and exposure during an early stage may in some cases be followed by serious results. The fact, however, that as a rule contagion is not active until the eruption has developed is an extremely important one. We have thus in scarlet fever a distinct advantage over measles, for in the latter

disease the period of contagion begins two or three days before the appearance of the eruption.

The question of sending the other children away from home is often a serious one. The decision must rest largely on the time of the exposure. If the exposure occurred before the appearance of the eruption, there may be little fear that the disease has been contracted. If exposure occurred during the stage of eruption, the probability of illness will be very great. If the patient is isolated soon after the initial symptoms have appeared, other children in the family are very unlikely to have taken the disease from him.

Whatever may be thought of the propriety of isolation during the period of incubation, there can be no doubt of its importance after the first symptoms have appeared. It should be complete with no relaxation. The patient is dangerous to others as long as the slightest desquamation continues on any portion of the skin. The duration of this period is extremely variable, and the most common error, perhaps, consists in being guided by a fixed number of days. The conventional forty days is to be regarded as only approximate. It is rarely too long. Desquamation is liable to persist in small areas of the body after its disappearance from other portions. These circumscribed areas are most frequently found about the flexures of the joints and about the finger-nails after desquamation has disappeared from every other part of the body. There can be no more dangerous place for such persistence, for the scales are liable to fall on any article which the fingers may touch and may hence be conveyed to a distance. There are many authentic cases of conveyance of the disease through letters written by desquamating hands. I have known of such an occurrence, the letter going several hundred miles through the mails. The hands and fingers should be particularly scrutinized before the quarantine is raised.

The subject of secondary and tertiary desquamation is interesting from the standpoint of prophylaxis. The scales from these desquamations are certainly less infectious than those of the primary desquamation. It is the belief of some observers that they are not capable of conveying the disease. There seems to be authentic evidence, however, that even in tertiary desquamations the scales have been infective and it is the part of wisdom, therefore, to regard every such case as unsafe.

Desquamation is not the only factor by which the period of isolation is to be determined. Purulent discharges contain the infective principle of scarlet fever. No child who is still suffering from otitis, chronic pharyngitis, or a purulent discharge of any kind should be allowed to mingle with others. These dangers have not been sufficiently recognized and as a result the disease has undoubtedly been communicated to many children. In many cases six weeks is ample quarantine and the patient may be released with perfect safety to others. In other cases he is almost as dangerous at this time as during the first week of the eruption. The rule for quarantine should be not a fixed number of days or weeks, but the time that is necessary for the disappearance of all desquamation and every purulent discharge. The question of infection after release



from quarantine has of late been the subject of some discussion in England, and an attempt has been made to prevent more fully what they call "return cases." At the Monsall Fever Hospital in Manchester, for example, the following method has been adopted: Certain wooden pavilions are set apart as convalescent wards for scarlet fever cases, no case being sent into these wards until six weeks have elapsed from the onset of the fever. Even then desquamation, as well as all purulent discharges, must have ceased. The convalescent children are encouraged to take exercise freely in the open air. I am unable to say how long they are retained in these wards, but the statement is made that since this system has been in operation there has been no "return case," although they were not uncommon before that time.

The question of isolating mild but undoubted cases of scarlet fever is frequently a trying one. In some of these desquamation is very slight and there is no purulent discharge. It is undoubtedly a fact that the quarantine can be raised in a few of these cases in less than six weeks. Still the desquamation, even when slight, is liable to persist for a greater period than its intensity might lead one to expect, and mild cases require more than ordinary precaution.

Schools and public assemblages are active agents in the dissemination of the infectious diseases. One means by which schools aid in the spread of scarlet fever is through the clothing of children who may have come from families where the disease exists. No better incubator for bacteria could be provided than some of the dark, close, warm school closets filled with damp clothing. The advisability of closing schools in an epidemic of scarlet fever must be settled differently in different communities. In the country and small towns, where the children will be separated from each other when the schools are closed, their closure may be an important measure of prevention. Moreover, in such communities people are known to each other. Illness is at once known and contagion can be guarded against. In large cities, on the other hand, the conditions are quite different, particularly in the crowded tenement regions. Here the children cannot and will not be confined to their homes, but will mingle with each other all day long. Closing of the schools will not prevent it. The daily inspection of the children in city schools is a great safeguard against the spread of the disease.

It is unquestionably a fact that medical men through carelessness have sometimes been instrumental in carrying scarlet fever. No other disease is so frequently transmitted through the agency of clothing. Sputa coughed out during examination or scales adhering to the clothes may cause the disease in other children upon whom the doctor may be in attendance. A practitioner should never visit a case of scarlet fever or diphtheria without a gown. Such a gown should be made to button closely about the neck and wrists, and should be long enough to reach to the feet and have a hood to cover the head. It should be put on before entering the sick-room and should be hung in the bath-room or other suitable place upon leaving. Upon the termination of the case the gown may be thoroughly boiled and used again.



In considerable experience, I have never found a parent or child who objected to it. On the contrary, it inspires confidence in the physician and removes the possibility of his ever being charged with bringing the disease into the house should its occurrence in one of his families be unexplainable. The hands and face should be disinfected after every visit to a scarlet fever patient. The same is true of the stethoscope, which should be used for all physical examinations of the chest. The tongue depressor or other instrument used about the throat should not be taken from the room. The doctor himself should not carry out the details of the treatment farther than is strictly necessary. It is difficult for him to do so without danger of carrying the disease. It can only be avoided by more thorough disinfection than most doctors are willing to undergo. In few diseases is the importance of a qualified nurse greater than in scarlet fever. One should be secured wherever it is possible. Not only are the details of prevention and treatment carried out more thoroughly and satisfactorily, but the doctor is relieved of many duties which he should not be obliged personally to perform.

*Sick-room.*—A room for the patient should be selected which can be most readily isolated and will at the same time be convenient and habitable. Six weeks' confinement to a single room is a trying ordeal, and isolation during the last days of the period can be more strictly enforced if the room is cheerful and comfortable. While a room at the top of the house is the most desirable, another should be selected if it is nearer to a bath-room. The passing to and fro to the bath-room will frequently undo all efforts at isolation, not to speak of the additional labor involved. All unused doors should be sealed with strips of paper or rubber plaster. The floor should be without carpet and if the boards are separated by wide cracks muslin should be tacked down. The ease of preventing the spread of the disease is greatly augmented if a third person is available. To this person is assigned the duty of carrying the food and various articles required by the nurse and of taking away the soiled clothing and performing the numerous offices outside the sick-room. She thus comes in direct contact neither with the sick nor the well. It is a hardship for the mother to make a choice between the invalid and the other children, particularly if they be small, but the necessity of her doing so is urgent. Such toys as are left for the child's amusement should never be removed from the room. Hanging dampened sheets before the doors is of some practical value. It is not to be supposed that they can disinfect the air or destroy the germs, but they do prevent currents of air when the doors are opened and are a constant reminder of the necessity of care.

The preparation of one room for a sick-room in a house where there are children is a wise measure. Such a room is not infrequently found in modern houses and should be more common. It may be made as cheerful and available for ordinary use as any other room. The walls and ceilings should be painted or covered with tiles or washable paper and the floor polished and covered with rugs instead of a carpet. The

hangings should be easily removable and the furniture should be plainly made of polished wood or white enamel. A room thus arranged can be quickly put into commission as a sick-room and will greatly simplify the question of prophylaxis.

General inunction of the body is a most effective measure both of treatment and prophylaxis. It may be begun as soon as the eruption has appeared and should be continued until desquamation has ceased. During the stage of eruption before the stage of desquamation has begun, a simple bland oil is most desirable. Antiseptics can be of little avail and all irritating preparations should be avoided. Lanolin is one of the best of these, or a mixture of equal parts of lanolin and cold cream. These preparations, however, are somewhat expensive. Vaseline, therefore, may be employed in their stead, and is no doubt the most common preparation used. Some of the cheaper grades of yellow vaselin are irritating to delicate skins. When itching and irritation of the skin is great, a 5 per cent. ointment of boric acid and vaselin is sometimes effective. Sponging with a solution of borax and water, followed by carbolized vaselin, will also give temporary relief. Carbolized vaselin, however, should not be used over large areas. In scarlet fever we do not have a healthy skin, and it seems quite possible that absorption might occur. In a disease in which the kidneys are frequently involved, it is unwise to run any risk of introducing so irritating a substance as carbolic acid.

After desquamation has begun the objects of inunction are quite different. The procedure becomes then a matter of prevention as well as treatment and the most important object is to soften and loosen the scales, thus preventing their dissemination with the resulting danger of spreading the infection. It seems somewhat doubtful that the scales can be disinfected by adding disinfectants to the oily substance used for inunction. But this must be settled at some future time by the bacteriologist. The general fact is at least positively settled that proper care of the skin during the state of desquamation is one of the most effective means of limiting the spread of the disease, and also without doubt of shortening the time of desquamation. Antiseptics may be added to the ointment or oil used for inunction. The boric acid ointment already referred to is one of the best. A 2 per cent. ichthyol ointment has its advantages, but is objectionable to many patients because of the odor. Carbolic ointment may be used over limited areas. During this stage the inunction may be preceded by a bath or sponging with water at a temperature of 90° F. The water may be plain or it may contain a small amount of salt or borax. The use of an antiseptic soap is advocated by many practitioners, resorcin soap being, perhaps, the most commonly used.

A preparation having even a slight odor becomes perceptible when applied to the whole surface of the body, and may cause loss of appetite. This is the chief objection to be urged against the animal fats like lamb fat, mutton tallow, or beef suet. The bacon rind popular in some parts of the country has the same objection and possesses no

advantages. Lard has but little odor, but it is difficult to obtain it pure in cities. The physician should always make sure as to the character of the preparation used for inunction. A rancid fat in a severe case of scarlet fever may cause great irritation and prolong the period of desquamation.

The best disinfecting agents for house use are bichloride of mercury and carbolic acid. A standard solution of bichloride of the strength of 1 : 1000 may be made by using "antiseptic tablets" or by dissolving 4 gm. (1 dr.) of bichloride and 30 gm. (one ounce) of common salt in 4000 c.c. (one gallon) of water. A standard solution of carbolic acid of 5 per cent. strength (1 to 20) may be made by dissolving 180 gm. (six ounces) of carbolic acid in 4000 c.c. (one gallon) of water. For the various conditions in scarlet fever and other diseases requiring disinfections, these solutions may be used as follows: For the hands and person the carbolic solution in one-half or one-third strength. For clothing, towels, and bedding the carbolic solution in full strength for one hour, after which they should be boiled. For closets, drains, and sinks, either solution in full strength. All discharges from the mouth and nose should be received in glass or porcelain vessels. Either solution should then be added in full strength and at least twice the volume of the discharge. After standing for one hour the whole may be thrown into the closet. For sputa cups, full-strength carbolic solution should be used. Certain dishes should be reserved for the sole use of the patient. They should be disinfected with full-strength carbolic solution and then boiled and rinsed. The remains of meals should be burned. When the patient has recovered, the entire body should be bathed and the hair washed with hot water and soap. He should then be dressed in clean clothes (which have not been in the room during his sickness) and removed from the room. The bodies of those who have died from scarlet fever or other contagious disease should be wrapped in cloths saturated with either solution, preferably the bichloride, in full strength. All the antiseptics named in strong solution are more or less irritating to the skin. For use about the eyes and other places a saturated solution of boric acid is largely used. It is not poisonous or irritating to the mucous membranes.

Prolonged boiling is one of the best antiseptic measures at our command. Hence, towels, handkerchiefs, and all articles of clothing and bedding which may be boiled or steamed can be thus sterilized. Handkerchiefs and towels should be used about contagious cases as little as possible. In their place pieces of old cloth or squares of cheese-cloth should be used, and these may then be burned, thus avoiding the trouble and possible danger from imperfect disinfection. If they cannot be at once burned, they should be at once dropped into one of the full-strength solutions. If the floor of the sick-room be bare, it should be wiped daily with the solution of bichloride in full strength. If the carpet is covered with muslin, this should be brushed over daily with the same solution.

At the termination of such diseases as scarlet fever, diphtheria, and

smallpox, all toys and books should be destroyed. Books are particularly dangerous, for they cannot be adequately disinfected. The room should be washed—floors, walls, and ceiling—with a full-strength bichloride solution, and the furniture should be wiped with the same antiseptic. Carpets, upholstery, hangings, bedding, and mattresses should, if possible, be disinfected with steam. When this is impossible, they should be wiped thoroughly with cloths dampened in the bichloride solution and then fumigated. After this they should be hung for days in the open air and sunlight. As it is difficult to certainly disinfect articles of this character except by steam, all those of lesser value should be sacrificed.

Before the sick-room is again occupied, it should be thoroughly fumigated. Fumigation with sulphur, as it is ordinarily done, is ineffective, owing to the small amount of sulphur used and the dryness of the atmosphere. The various objects in the room should be dampened, and steam should be generated in it if possible. Three pounds of sulphur are necessary for each 1000 cubic feet of air space, with eight hours' exposure. The sulphur is best used in the form of fumigating candles, which may be found in every drug-store. It is best to place each candle in a shallow basin of water to avoid danger of fire. The room should be sealed by pasting strips of paper or rubber plaster over all cracks and key-holes. It should be kept closed for at least eight hours after the sulphur is lighted and thoroughly aired before it is again occupied.

Formaldehyde gas is superior to sulphur for room fumigating. It is commonly generated from formalin, which is a solution of formaldehyde in water. For this purpose several generators have been devised. Not less than 175 c.c. (six ounces) of formalin should be used for each 1000 cubic feet of space, and infected articles should be exposed to its action for not less than four hours. Formaldehyde burns easily, and may be set on fire by an open flame. It is an excellent deodorizer as well as disinfectant. The necessary apparatus is now in the hands of most boards of health, and a small generator sufficient for the disinfection of rooms of ordinary size can be obtained at not large expense. In the absence of a generator the formalin may be evaporated from sheets suspended from the ceiling. It is very irritating and must be handled with care.

It must be remembered that in scarlet fever we have not the sure basis of knowledge which we possess in diphtheria. Until a specific germ has been discovered and its life history studied, we must rely on clinical evidence alone. We must, therefore, expect to find differences of opinion on almost every detail of pathogenesis, prophylaxis, and treatment.

**Treatment.**—Scarlet fever is still a disease over which we have but little direct control. Many specifics have been proposed, tried, and found wanting. Much may be done to avert complications and render them less serious when they occur, and many lives may be saved by judicious management. Mild cases require little or no medication; they usually receive too much. The disease is self-limiting and while



it is running a normal course more harm than good will result from vigorous treatment. There are times, on the other hand, when treatment of the most vigorous nature is necessary to save the life of the child. The physician should see that the patient is not kept too warm. Fear of cold and dread of water in the eruptive diseases must constantly be combated. It is not necessary, but rather harmful, to sweat the patient to "bring out the rash." The popular fear of bathing in the eruptive fevers has no rational foundation.

The patient should be kept in bed for at least three weeks. In complicated and prolonged cases the rule should be that the child should not be allowed to leave the bed for at least a week after the fever has subsided. It is exertion and chilling of the body which render late complications of mild cases so common. It is the best rule, therefore, to keep every child ill with scarlet fever confined to the bed for twenty days, even if the attack be very mild. Quiet in bed and a liquid diet will do more to prevent the late complications than any other means at our command. If the rule is laid down at once that the patient is to remain in bed for three weeks it can always be carried out. Patients will usually accept the inevitable with but little objection, but will become restless under uncertainty or half-hearted methods.

Milk is the best diet for scarlet fever patients. It may be given peptonized or plain. If milk is disliked by the patient, kumyss, zoalak, buttermilk, or junket may perhaps be substituted. If these preparations are not taken well, gruels or foods made of rice, arrowroot, cornstarch, farina, barley, or wheat flour may be available. Animal broths may be given sparingly to form a variety. Barley-water flavored with mutton or chicken-broth is an excellent substitute if milk becomes too irksome. Cocoa or chocolate may also be used to cover the taste of milk. It may be given two or three times a day with hot milk. Plain vanilla ice-cream may be given in small amounts when the throat is dry and sore. Milk should be used exclusively if possible during the first two weeks and should form a large part of the diet during the subsequent four weeks. Such a diet with rest in bed will do much to prevent renal complications. Water should be given freely during the whole course of the disease. It aids in eliminating waste products from the body and perhaps the scarlatinal poison, and thus diminishes renal irritation. If signs of nephritis appear, all other food should be at once stopped and the patient should be placed again on a milk diet and water should be given freely. Nitrogenous food should be used sparingly for two months and meat should be wholly eliminated from the diet for that length of time. As the patient becomes convalescent the diet may be increased by the addition of milk-toast, junket, plain rice-pudding, cornstarch, custards, crackers, cereals, animal jellies, baked apples, and stewed fruits. In the later weeks eggs, oysters, fish, and chicken may be given.

The initial vomiting usually requires no treatment, but the bowels should be acted upon mildly by small, repeated doses of calomel. Later they should be kept acting, if possible, by means of enemata rather than

by the use of cathartic drugs. If the vomiting is persistent, food should be withheld for ten or twelve hours and hot water or cracked ice should be given.

While the eruption is developing and is at its height the itching and burning of the skin are sometimes very distressing. These may be mitigated by the use of a weak wash of carbolic acid and borax or by the use of carbolized vaselin. In some cases relief is obtained by sponging with a solution of bicarbonate of soda in water (a level teaspoonful to a quart), followed by anointing with cold cream. In other cases simple talcum powder gives more relief. Bathing the surface with warm water followed by anointing with plain or carbolic vaselin or some bland ointment should be begun as soon as the first signs of desquamation appear and should be continued throughout the course of the disease. This daily rubbing of the surface with oil is a most important measure of treatment and should never be omitted. As to the oily substance used, my own preference is for cold cream. When well made, it is never irritating and does not remain on the skin or soil the clothing as do many oils. White vaselin and pure lard are, however, largely used and are less expensive than cold cream. Other details of injunctions have already been given.

In mild cases stimulants are not required and are rarely necessary in cases of ordinary severity. In severe cases they are frequently required for a few days and in some instances must be used persistently and freely. Alcoholic stimulants are the first to be selected. They are required in all the septic cases as well as those of the malignant type. As in other conditions, digitalis is indicated when the pulse becomes soft and weak and of low tension. Holt gives one minim of the fluid extract four times a day at five years. Owing to the tendency to renal and cardiac complications, digitalis is a drug of especial value in scarlet fever. Strychnine is also of value in septic cases with prostration. At five years of age 0.00032 gm. ( $\frac{1}{2000}$  gr.) may be given.

In ordinary cases antipyretic treatment is not necessary, but in other cases the temperature may require attention from the outset. It should not be forgotten that a high temperature is normal to scarlet fever. It may be allowed to run, therefore, without interference, to a somewhat higher point than in most other diseases. Hyperpyrexia or a temperature continuously above 104° F. demands treatment. It is best reduced by means of the cold bath; but this for obvious reasons is less practical in private than in hospital practice. The cold pack or cold sponging is more available. An effective method of applying cold adopted at the Willard Parker Hospital is thus described by Northrup: "The tendency in all cooling processes is for the feet to become cold. To obviate this the patient is placed upon blankets, but the legs, feet, arms, and hands are wrapped in warm, dry blankets and hot bottles are enclosed in the wrappings. An ice-bag is applied to the head. The face and head are freely sponged in warm water and alcohol, evaporation being hastened by fanning so long as it cools the patient, clears the cerebrum, gives force and improved rhythm to the heart, and leaves the patient to

quiet sleep." Great caution should be exercised in the use of antipyretic drugs. The coal-tar antipyretics are capable of doing much harm if injudiciously administered. Tepid sponging with ice to the head is usually effective in mitigating the less pronounced nervous symptoms. Opiates are rarely to be advised. The coal-tar products are not to be used as antipyretics, but phenacetin in small doses is admissible when there is extreme restlessness and the child is losing strength from sleeplessness. For the convulsions which occur in rare instances in septic cases, warm baths and chloral administered by the rectum 0.324 gm. (5 gr.) at five years should be employed.

Burning and soreness of the throat during the first few days may be mitigated by giving cool water or bits of ice. In the simpler forms of pharyngitis, hot drinks may be given or irrigation of the back of the throat with hot saline or boric acid solutions, about 4 gm. to 475 c.c. (one drachm to a pint) may be employed. Chlorate of potassium should be avoided. Its beneficial effects are doubtful and its known irritating effect upon the kidneys contraindicates its use. Nasal irrigation should be avoided unless clearly indicated. Jackson, of the Boston City Hospital, has seen less otitis when it has not been generally employed. Irrigation is indicated by a purulent nasal discharge or obstruction of the nasopharynx. More harm than good may result from overzealous attempts at local treatment of the throat and nose. Peroxide of hydrogen is, in my opinion, an unsafe remedy in such conditions. It is an irritant even when rendered alkaline, and it has the power to prolong indefinitely the presence of pseudomembrane. The most successful treatment consists not in the use of active and poisonous antiseptics, but of mild and cleansing washes freely and frequently applied. The error should not be made, on the other hand, of failing to irrigate the nasal passages when seriously obstructed either by a purulent or by a thick, tenacious discharge. It is especially essential if adenitis be present or is increasing. The solutions used for this purpose should always be warm.

Adenitis can only be controlled by checking the septic process in the throat. The application of hot oil or the hot-water bag is soothing to some patients, but the use of cold is preferable in most cases. Small ice-bags applied to either side of the throat usually give comfort to the patient and have some controlling effect upon the swelling. A long, thin ice-bag tied by a string in the centre to form two sections is more easily kept in place than are two smaller ones. Poultices should be applied for short intervals only. Their continuous use renders the parts sodden, favors suppuration, and after a time increases the pain. An ointment of ichthyol and camphor is a favorite mode of treatment with some practitioners; 4 gm. (1 dr.) of ichthyol and 0.65 gm. (10 gr.) of powdered camphor may be used to 31 gm. (1 oz.) of ointment. Suppuration should be treated by free and early incision.

Otitis requires the treatment demanded by the disease in other conditions. Early puncture of the drum membrane removes a part of the danger of extension to the mastoid cells. The joint affections of



the ordinary type require but little treatment aside from rest and protection. The joints should be wrapped in absorbent cotton or bandaged with flannel. If the sleep is broken by pain a mild opiate for one or two nights is admissible.

*Nephritis* should receive prompt and very careful treatment. Frequent examinations of the urine should be made and treatment should begin promptly upon the first appearance of albumin after the second week. It should be remembered that it is an especially acute nephritis which is present and that all irritating drugs should be carefully avoided. The saline diuretics like citrate and acetate of potash are especially helpful. *Digitalis* is of peculiar value in this form of disease. A freshly prepared infusion is the best preparation for such use and may be given at the outset in doses of 4 c.c. (1 dr.) every four hours to a child of five years. It may be combined with a saline diuretic. The free administration of water throughout the course of the disease is important as a preventive measure as well as a measure of treatment. Lithia water is, perhaps, more helpful than plain water. Flushing the bowel with hot water after the method of Kemp with a double-flow rectal tube, is another measure of especial value. A little albumin may appear for a few days without symptoms of any kind and may soon disappear without leading to serious consequences. It should, however, always be heeded as a danger signal. A milk diet should be given and the patient should be carefully watched.

The serum treatment of scarlet fever has received very careful study from some of the ablest observers. It can only be said that it has proved disappointing. The last reports at the time of writing are distinctly unfavorable. Baginsky, who has been a champion of the idea that scarlet fever is the result of streptococcus infection, has very recently reported his results with the serum treatment. He first used the Marmorek antistreptococcus serum and later the Aronson serum. He reports a series of 62 cases treated with the latter. The mortality among these cases was a little lower than that among a series of cases treated without it, but the difference was so small as to offer but little ground for encouragement. Neither the general condition nor any particular symptom was materially changed for the better. Because of its apparent unfavorable action in 4 cases the use of the serum was not continued. Escherich has also reported results with another form of serum, but without any material improvement in the mortality rates. When given early and in large quantities there was some apparent beneficial effect. From all the evidence available the serum treatment is not to be commended.

As emaciation and anemia are frequent results of scarlet fever, active tonic treatment should be instituted during convalescence, the chief reliance being placed upon iron. Basham's mixture is especially indicated. The patient should be carefully protected from cold, for exposure not infrequently seems to precipitate nephritis long after the period of its usual occurrence. The urine should be examined at intervals after the child has fully recovered and the tonic treatment should be continued for a considerable time if the anemia persists.



## CHAPTER XX.

### MEASLES—RUBELLA—FOURTH DISEASE—ERYTHEMA INFECTIOSUM.

#### MEASLES.

By FLOYD M. CRANDALL, M.D.

MEASLES, Rubeola, or Morbilli, is an acute, infectious, and contagious disease occurring most commonly in children. Typical cases present the following features: After an incubation of twelve days there is a gradual invasion marked by fever with dry, metallic, teasing cough, coryza, and suffusion of the eyes, followed on the fourth day by a coarse, maculopapular eruption which appears first on the temples, neck, and sides of the face. The eruption spreads slowly until the body is covered, and appears last on the hands and feet. It continues for about five days and slowly fades away in the order in which it came. It is followed by a bran-like desquamation, which usually continues not longer than seven or eight days. Measles is contagious from the first symptoms of coryza, a fact which partially explains its widespread occurrence. Susceptibility to measles is greater than to most other diseases and very few escape it.

**Etiology. Exciting Cause.**—Measles is the most contagious of the infectious diseases except smallpox, but the infective principle soon disappears from rooms and clothing. No specific micro-organism, however, has been discovered. Whatever the exciting cause may be, it is evident that it is very diffusible and of low vitality.

**Predisposing Causes.**—Predisposition to measles is more universal than to any other disease except possibly smallpox and influenza. Every child over one year of age who has not already had it may be expected to contract it upon exposure. Adults who have not had the disease are also more susceptible to it than to the other infectious diseases. Under one year measles is rare and under six months is extremely infrequent. I have seen an infant of six months who was kept in a room with a measles patient during the whole course of the disease without contracting it. Cases have been reported, however, of newborn infants contracting the disease from their mothers who were suffering from it at the time of birth. I have recently seen a typical case in an infant of five months. The comparative immunity of adults is explained largely by the fact that few escape infection during childhood. In localities where the disease has not prevailed for years it has been noted that all ages and conditions suffer. Sex is not a predisposing factor and has no relation whatever to the occurrence of the disease.

Neither is social condition a predisposing factor, for children living in hygienic surroundings are apparently as susceptible as those living in tenement districts.

Measles is endemic in all cities and large towns, but at intervals becomes epidemic and spreads over a wide area before it expends itself. These epidemics are frequently widespread and affect large numbers of children. There is no law of periodicity governing epidemics of measles. They are more common during the colder months of the year and are rare during the summer. In New York City the disease is most common during the late winter and early spring and is least frequent in the early autumn. Notwithstanding the fact of the great susceptibility shown by most children to measles, some are occasionally seen who appear to be immune. They do not contract the disease after prolonged close exposure. I have recently seen a marked case of this character.

*Sources of Infection.*—Measles may be transmitted by direct contact, and, hence, is a true contagious disease. The area of contagion is large and very brief exposure is sufficient. It may be conveyed a considerable distance through the open air. In an enclosed room it may be contracted by a child fifteen or twenty feet from the patient. It seems possible that the contagium may be conveyed by the breath, but it is certain that it resides in the sputa and the discharges from the nose and eyes. It has been conveyed to monkeys by inoculating their throats with mucus obtained from the throats of measles patients. It is probable also that it resides in the desquamation scales, but is far less potent than is the poison carried by the desquamation of scarlet fever. The disease may be conveyed by clothing or bedding, or it may be contracted by a susceptible person entering a room which has recently been left by a measles patient. At the Randall's Island Hospital measles was conveyed by a kitten which escaped from a measles ward and was allowed to lie in the bed of several children in another ward. Such intermediate contagion, however, is very rare in measles. It is doubtful whether it is ever conveyed from one child to another by a person who has been only for a short time in contact with a patient. It seems entirely possible that a nurse who goes directly from a measles patient to a healthy child without disinfection might transmit the disease if she comes also in close contact with it. I am not, however, personally aware of such a case. The case of the cat is the only one of undoubted intermediate infection which has come under my own observation.

*Period of Incubation.*—The period of incubation ranges from nine to twenty-one days. It was found by Holt to be between eleven and fourteen days in 66 per cent. of 144 carefully observed cases. In but one case was it less than a week. From all the evidence available I should give twelve days as the most common period of incubation. The average period differs somewhat in different epidemics, being a little shorter some years than others.

*Period of Infection.*—Measles is contagious from the first appearance of the catarrhal symptoms. Well-authenticated cases are recorded in which it was transmitted four days before the rash appeared. It is

believed to be most contagious when the fever and catarrhal symptoms are at the highest. The contagiousness diminishes as the active symptoms subside and is slight during the stage of desquamation. Except in complicated cases in which the catarrhal symptoms are prolonged or purulent discharges are present, the contagious period is not, at the longest, over twenty-eight days. In most cases it is passed at the end of twenty-one days. It is proper to add that there are still differences of opinion regarding the period of greatest contagiousness, the belief of some being that it is actively contagious during desquamation.

**Pathology.**—The lesions of measles are confined to the skin and the mucous membranes of the conjunctiva, nose, pharynx, larynx, and the larger bronchial tubes. The changes of the mucous membranes are as much a part of the disease as are those of the skin. The morbid changes of the skin are those of hyperemia. On the mucous membranes they are those of acute catarrh. Pseudomembranous inflammation may occur in complicated cases. The complications are apparently due to other micro-organisms than the specific germ of measles. Complicated measles is, therefore, a mixed infection, the most common complicating germ being the staphylococcus. The streptococcus is, however, often present and, as a rule, causes more serious lesions than does the staphylococcus. The pneumococcus is also frequently found. As pneumococci and streptococci are frequently present in hospital wards, measles occurring in hospitals is very prone to be complicated. In an epidemic in the Infants' Hospital, Randall's Island, the first 12 cases which developed were complicated by pneumonia. In children's hospitals this tendency to complication by extraneous germs renders measles one of the most dreaded of diseases, the death rate often being very high.

**Clinical Types.**—Measles, as a rule, presents less variation from the classical type than does scarlet fever and most of the other infectious diseases. Very mild cases sometimes occur, but they are less common than very mild cases of scarlet fever, while, on the other hand, malignant cases are also less common. Measles is also fairly constant in its duration and the various stages are well defined. Although the type of disease which I have designated as the ordinary type is most common, measles is capable of occurring in very irregular and atypical forms. Such irregular types occur most commonly in children under three years. In most epidemics a larger proportion of measles cases will run a regular course than will a similar number of cases of scarlet fever, but in some epidemics unusual types may be repeated again and again. Reports of certain epidemics, therefore, not infrequently show a far greater number of complicated or irregular cases than the averages based on the experience of several years. Thus, in an epidemic of 423 cases occurring in Canada, as reported by C. J. Edgar, over 200 were of hemorrhagic form and 103 were classed as malignant.

**Ordinary Type.**—The onset of measles is usually gradual and is characterized by feverishness, sneezing, coryza, suffusion of the eyes, photophobia, and a general feeling of illness. Occasionally a chill

followed by a high temperature is the initial symptom. Within twenty-four hours after the advent of the first symptoms a characteristic, hard, dry cough appears and the child shows all the signs of a catarrhal cold. The coryza, however, is more marked than is that of an ordinary cold and the cough has a peculiar metallic character. The fever increases as the eruption appears and frequently is at its height on the first day of the eruption. A few spots commonly appear on the afternoon of the fourth day, but may sometimes be seen as early as the second day and in rare cases as late as the fifth or sixth day. The early appearance of the eruption is more common in young children. There are no characteristic constitutional symptoms upon which a diagnosis can be made.

The temperature on the first day is usually not above 102° F. but will occasionally be found at 103° or 104° F. The fever does not, as a rule, range as high in measles as in scarlet fever. After a sharp rise on the first day, the temperature not infrequently falls on the second and third days, but increases as the eruption begins to appear and reaches its height on the second day of the eruption. It then falls gradually day by day and becomes normal between the seventh and ninth day of the disease. Not infrequently there is a sudden fall on the sixth or seventh day, forming almost a crisis. The diminution of the fever on the second or third day is sometimes so decided as to lead to error in diagnosis. The possibility of such a fall should not be forgotten, particularly should the catarrhal symptoms and cough continue undiminished. The fever and constitutional symptoms are usually at their height when the eruption has reached its fullest development, on the fourth or fifth day of the disease.

The eruption, as already stated, more commonly appears on the fourth day. It is first seen on the temples and sides of the face or on the neck and behind the ears. At first it generally consists of small red spots having no strictly characteristic appearance. They rapidly increase in size and form small macules or very slightly elevated papules on a slightly reddened base with normal skin between. They are crescentic or circular in shape, and, being hyperemic in character, disappear on pressure. The eruption as it develops becomes confluent in places, particularly on the face, where it assumes a blotched appearance. The eruption usually reaches its height at its first site of appearance at the end of thirty-six hours; it remains stationary for about two days and then rapidly fades away. It extends over the body somewhat slowly, appearing on the trunk and limbs on the second day. The wrists and backs of the hands are commonly the last to be involved. When at its height in these places, the rash has sometimes partially faded on the face and neck. The spread of the eruption is sometimes extremely rapid, the whole body being covered in a few hours, but this is rare. Desquamation begins as soon as the eruption has faded, and follows the order of its appearance. It rarely continues more than ten days in any given area and may be of much shorter duration. It is most intense where the eruption has been most intense. It occurs in branny patches



## PLATE XVI

Fig. 1



Fig. 2.



Fig. 3



Fig. 4.



### The Pathognomonic Sign of Measles (Koplik's Spots).

FIG. 1. The discrete measles spots on the buccal or labial mucous membrane, showing the isolated bluish-white spot with the minute bluish-white centre on the normally colored mucous membrane.

FIG. 2. Shows the variety of diffuse eruptions on the mucous membrane of the cheeks and lips: patches of pale pink interspersed among confluent patches, the latter showing numerous pale bluish-white spots.

FIG. 3. The appearance of the buccal or labial mucous membrane when the measles spots are completely coalescent and give a diffuse redness with the appearance of bluish-white specks. The exanthema on the skin is at this time generally fully developed.

FIG. 4. Aphthous stomatitis apt to be mistaken for measles spots. Maculae palatae puncta are surrounded by a red area. Always discrete.

1

1  
1  
2  
2

quite unlike the lamellar desquamation of scarlet fever. It is often so slight as to be completely overlooked, particularly when inunctions of the skin have been carefully used. Desquamation is usually completed within twenty days after the onset of the disease.

A few years ago a symptom was described by Koplik, of New York, which has been accepted as a valuable aid in the early diagnosis of measles. This symptom consists in the appearance of a certain characteristic eruption on the inside of the cheeks and lips. On the first day of the invasion the examination of the buccal mucous membrane in a good light will reveal a scattered eruption consisting of small, irregular spots of bright-red color, in the centre of each of which is a minute, bluish-white speck. These spots are now regarded by most authorities as pathognomonic of measles. They are easily overlooked by one not familiar with them, and too much reliance should not be placed on this symptom by the average practitioner. On the other hand, other conditions may be mistaken for Koplik's spots by the inexperienced.

The constitutional symptoms of measles, while somewhat variable, are fairly characteristic. They are at their height during the stage of eruption and are usually most intense on the fifth or sixth day of the disease. The fever then abates and all the symptoms begin to subside. This sometimes occurs on the sixth or seventh day so suddenly as to form a crisis. This, however, is not the rule. When the disease is fully developed the patient presents a striking appearance. The face is covered by a blotchy or confluent eruption and is swollen and edematous; the eyes are red and sensitive to the light and are filled with a mucous or mucopurulent secretion; the nose is swollen and discharges a similar secretion; there is a dry, metallic, and very troublesome cough; the tongue is coated; the appetite is completely lost; the bowels are frequently relaxed; the child lies in a heavy and stupid condition, but is restless and irritable when disturbed; the lymph nodes at the angle of the jaw are frequently enlarged and not infrequently the postcervical lymph nodes also.

With the disappearance of the fever a change in the character of the cough occurs. It loses its metallic sound and harassing character and becomes looser and less irritating to the patient. It frequently disappears within a week, but sometimes evidences of bronchitis continue and the cough proves a troublesome symptom for several weeks. In most cases the photophobia subsides rapidly and the eyes become normal, but often remain weak and watery. If strong light is admitted too soon a mild but very troublesome form of conjunctivitis may result. Other symptoms usually subside rapidly; the child becomes brighter and less irritable; the appetite returns and evidences of illness soon disappear.

*Mild Type.*—This type presents no material variation from the usual type except that of mildness. The eruption is not well marked, the fever is slight, and all the symptoms are mild. The onset is sometimes of the usual nature, but the fever does not become high and the disease subsides rapidly. The catarrhal symptoms are sometimes slight and

some of the older authors laid considerable stress upon *morbilli sine catarrho*. A diagnosis of measles should be made with great hesitation when there is no corvza, suffusion of the eyes, or cough. Such cases have undoubtedly occurred, but they are exceedingly rare. A diagnosis can be made with certainty only with the knowledge of positive exposure.

*Severe Type.*—Measles sometimes appears in severe form even when there are no complications. The fever ranges unusually high, the eruption is intense, and the catarrhal symptoms are excessive. The child may be delirious, but more commonly lies in a comatose condition for a day or two. The disease does not greatly vary from the average type except in the severeness of all the symptoms, and may not be longer in duration than are the milder forms. Such cases always require very close attention. It should not be forgotten that a temperature which ranges unusually high is generally due to a complication. This is particularly true if the fever continues unabated as the eruption fades. The complication which most commonly causes an excessive or unduly prolonged fever is pneumonia. Any marked variation from the usual type demands particular attention, for it commonly indicates a complication. It is not safe to assume that it is a simple severe case until thorough examination has eliminated all possible complications.

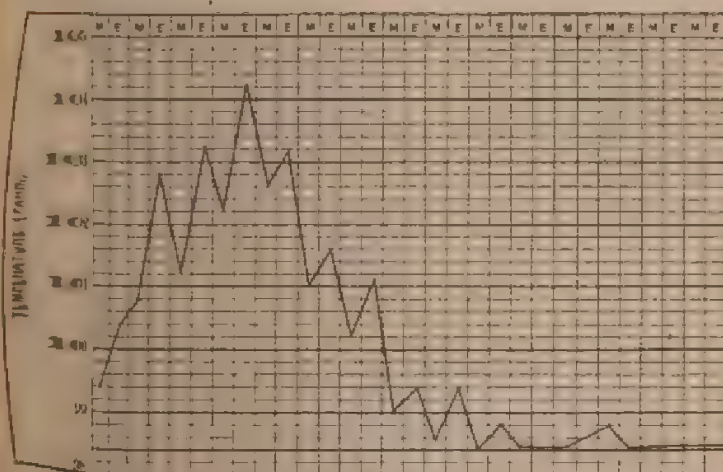
*Malignant Type.*—The malignant type of measles, marked by intense and overwhelming symptoms from the outset, is rarely seen outside of institutions. A type known as black measles is occasionally seen in certain epidemics. The name is derived from the color of the eruption, which is the result of hemorrhage. Small petechial spots take the place of the regular eruption. In many malignant cases the rash is faint or late in its appearance. As in scarlet fever, the system may be overwhelmed at the outset by the poison of the disease itself and the characteristic symptoms scarcely develop before death occurs. In others the disease seems to expend itself upon the lungs and the pulmonary symptoms develop at the outset. The diagnosis is at times difficult and sometimes would be impossible if the disease were not known to be prevalent. In my own experience the so-called malignant cases have often been, as a matter of fact, complicated cases. In an epidemic on Randall's Island, pneumonia would sometimes develop at the outset and consolidation could be detected before the appearance of the eruption. Carr had similar cases in the same hospital service. In such cases the eruption, instead of being intense, is often faint. There are, however, rare cases, as already stated, in which the patient is overwhelmed by the poison of the disease itself.

*Relapse and Recurrence.*—True relapse in measles is extremely rare. Its occurrence in fact is doubtful. A secondary rise in temperature after a normal fall sometimes occurs, but is almost invariably due to some complication. In such cases reappearance of the rash and recurrence of the catarrhal symptoms are not seen. In more than 700 cases of measles carefully observed by Conby not a single case of recurrence or relapse was seen. Second attacks of measles undoubtedly occur.



This is probably more common than in most of the other contagious diseases. It is not, however, as common as popular reports would lead one to suppose. It is extremely doubtful if three or four attacks of measles ever occur in the life of the same individual, though the doctor is constantly hearing of such cases. Rotheln is frequently mistaken for measles even by physicians, and many attacks due to disordered digestion are also called measles. I once attended a child whose parents insisted emphatically that he had had measles four times. Upon the appearance of the real disease of ordinary severity they were seriously alarmed because the attack was so radically different from any one of the others, and they were then in doubt as to the genuineness of the preceding attacks.

FIG. 113



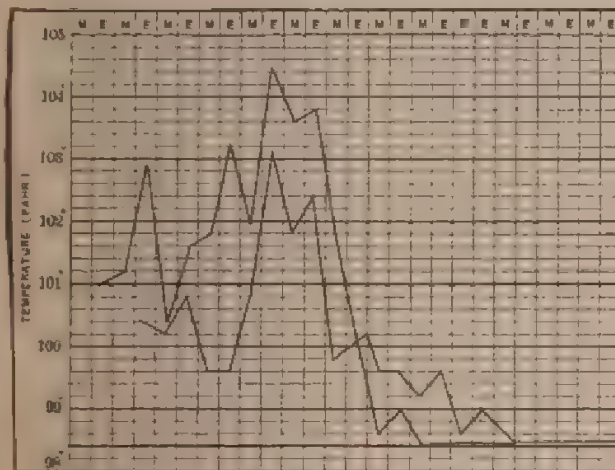
In non-polluted measles in a girl of five years, the rash appearing on the fourth day. This is the characteristic temperature curve of measles. It is characterized by a gradual rise and a gradual fall, the highest point being reached as the rash begins to appear, on the afternoon of the fourth day. After a "midway rise" for four afternoons. This chart is almost identical with charts presented as typical for measles by Holt and by Ashby and Wright.

**Symptomatology. Invasion.**—The invasion of measles is usually gradual, so much so in fact that it is often difficult to determine the exact time of onset, and the character of the disease may be indefinite before the catarrhal symptoms are present. The first symptoms are usually suffusion of the eyes, with acute coryza and general malaise. There is nothing characteristic about any of these symptoms, and unless exposure is known or expected there may be no suspicion that the child has more than an acute cold. The positive finding of Koplik's spots is a great help to an early diagnosis. In some instances the onset is abrupt, but an abrupt invasion with continuous high temperature is most often due to a complication, usually pneumonia. The period of invasion lasts commonly from three to four days. It is occasionally as short as one day and as long as five. Only in rare instances is it



*Temperature.*—As in most febrile diseases, the fever of measles sometimes pursues an atypical course. In the uncomplicated disease, however, a markedly atypical temperature range is not common. The most common temperature curve is one marked by a gradual rise for four or five days followed by a gradual fall, the temperature becoming normal between the seventh and ninth days. In an abrupt invasion, however, the temperature is sometimes found at  $102^{\circ}$  or  $103^{\circ}$  F., or even higher on the first day. In these cases of abrupt invasion the fever usually subsides on the second and third days, but rises rapidly as the rash begins to appear. A critical fall is also sometimes seen in the temperature curve of measles. This may occur at any time after the fourth day, but is most common on the sixth or seventh day. When

FIG. 116



temperature range of two cases of measles, the patients being brothers, aged five and ten years, placed at the same time and lying ill in the same room. The case of the younger was mild; that of the older was of the ordinary type. Though the older was taken ill twenty-four hours before the younger, the rash appeared simultaneously on the afternoon of the fifth day of the first case.

the fever of measles continues unabated after the end of a week or when it rises steadily after the appearance of the eruption, a complication should always be suspected and carefully sought for. It is true that the disease is occasionally prolonged and continues for several days after the eruption is at its height. As a rule, however, the fever begins to subside within a few hours after the eruption on the body has reached its full development (Figs. 113, 114, 115 and 116).

*Pulse.*—The pulse of measles shows nothing characteristic. The pulse curve is quite similar to the temperature curve and in the absence of complications increases and diminishes much as the fever rises and falls.

*Eruption.*—While the eruption in a large proportion of cases follows a typical course, it not infrequently departs from it. It usually requires

thirty-six hours to attain its maximum development on the face, but occasionally it requires twice that time. It not infrequently begins to fade on the face by the time it has made its appearance on the backs of the hands and tops of the feet. Unlike the eruption of some diseases, it frequently appears on the soles and palms. In most cases as the rash disappears a faint reddish or brown stain is left. This frequently disappears after two or three days, but may sometimes be seen after a week or ten days. This staining may be of aid in determining the nature of the disease when seen in a late stage. (See Plate XVII.)

One of the most serious departures from a normal rash is the hemorrhagic form known as black measles. Instead of the usual hyperemia form, small hemorrhages occur. These may be limited to small areas or may extend over the whole body. They indicate a severe form of the disease and warrant a grave prognosis, but this form is not as generally fatal as is popularly supposed. It is more commonly observed in hospitals and is rarely seen in private practice. On the other extreme from this condition is a very faint rash which is visible for but a day or two and sometimes only a few hours. In malignant cases the rash may be very faint or may not appear at all, but this is less common in measles than in scarlet fever. In very rare cases a fine rash may appear resembling that of scarlet fever more than the usual one of measles. The eruption of measles sometimes recedes suddenly. This usually results from failing heart power and poor circulation. The rash, it should be remembered, is hyperemic, and anything which seriously changes the peripheral circulation will modify the eruption. The popular belief that in such cases the rash "has struck in" is, of course, without foundation in fact.

*Desquamation.*—The desquamation of measles is very rarely profuse, as in scarlet fever. When the case is mild or when incisions are frequently performed the desquamation may be so faint as to be almost imperceptible. It is fine and branny and the skin does not strip off in shreds and scales as in scarlet fever. In many cases a peculiar and characteristic so-called "mousy" odor is present.

*Conjunctivitis.*—The eyes become red and watery very early in the course of the disease. This condition increases in severity until the eruption reaches its height and disappears rapidly as the fever subsides. There is usually an itching or burning sensation, but occasionally a child seems to suffer but little from the eyes. Recovery is usually complete, but occasionally chronic conjunctivitis, granulated lids, and other local conditions are left behind. The sight is rarely, if ever, impaired.

*Angina.*—An exudate on the tonsils or pharynx is not an essential part of measles. True diphtheria may occur as a complication, but is no necessary part of the disease. Catarrhal pharyngitis is an essential part of measles. Not only the pharynx but the uvula and tonsils are usually involved. They present on examination intense uniform redness, while over the hard palate numerous small red spots may be seen a little before the time of the eruption on the face.





Eruption of Measles.



**Complications and Sequelæ.**—The most common and serious complications of measles are bronchopneumonia, membranous laryngitis, otitis, and diarrhea; the most common sequelæ are tuberculosis and conjunctivitis.

**Pneumonia.**—Catarrh of the bronchial tubes is so constant an accompaniment of measles as to be classed as an essential part of the disease and one of its symptoms, but it is easy for the inflammation to extend from the smaller bronchi to the alveoli, thus transforming a normal condition into a most serious complication, namely, bronchopneumonia. The younger the child the greater this danger. It occurs chiefly in children under three years and is comparatively rare in children over four years. It is very common in institutions and renders measles the most dreaded of all epidemic diseases in infant hospitals, diphtheria being no exception to the rule.

In a recent epidemic of measles in the Infants' Hospital of New York, every case in children under eighteen months was complicated by bronchopneumonia or croup. The pneumonia usually made its appearance soon after the eruption reached its height, but developed in a few cases during the stage of invasion, the disease being regarded in two instances as simple bronchopneumonia until the eruption suddenly appeared. According to Holt, 10 per cent. of all cases are complicated by bronchopneumonia. He agrees with Henoch that a certain amount of pneumonia is found at autopsy in almost every fatal case.

Observations made by Méry showed that both the pneumococcus and streptococcus are present in the saliva of children ill with measles with much greater frequency than in health. This is one explanation, therefore, of the comparative frequency of pulmonary complications. Lobar pneumonia is an occasional complication of measles in children over four years, but is seldom, if ever, found under three years. Pleurisy or empyema is sometimes a sequel of such complicating lobar pneumonia. The signs and rational symptoms of either form of pneumonia complicating measles present nothing unusual.

**Pharyngitis and Laryngitis.**—While catarrhal pharyngitis is an essential symptom of measles, pseudomembranous pharyngitis occurs occasionally as a complication. Instead of invading the nose and ears as in scarlet fever, it shows a strong tendency to invade the larynx; but croup frequently develops without the appearance of membrane in the pharynx. The pseudomembranes which develop during the early stages or at the height of the fever are usually pseudodiphtheritic in character, being due to streptococci. Those which develop later are usually due to the Klebs-Loeffler bacilli and are true diphtheria. This secondary streptococcic disease, however, is quite as fatal as the bacillary disease. Not only is the child in imminent danger from laryngeal complications, but bronchopneumonia often develops as the direct result of streptococcic infection. The differential diagnosis between true and false diphtheria can rarely be made with certainty from clinical appearances alone. Fortunately, in private practice both complications are rare in children over four years.

*Otitis.*—While of less common occurrence than in scarlet fever, it sometimes occurs. It does not, however, usually prove so serious a cause such permanent damage. Suppuration is common and both are usually involved. The disease presents in its symptoms, however, nothing different from the usual course.

*Diarrhea.*—Complete anorexia is common during the febrile stage. Diarrhea is of frequent occurrence and may be so serious as to prove a grave complication. It may be due to simple intestinal indigestion or it may be the evidence of enterocolitis. It usually commences as fever is beginning to subside. If not checked, it increases in severity and may continue for days or weeks. It is more common in young children and is far more frequent in hospital than in private practice.

*Tuberculosis.*—The most common sequel of measles is tuberculosis. It commonly occurs as a tuberculous bronchopneumonia, general miliary tuberculosis, tuberculous adenitis, or tuberculous joint disease. It may result from primary infection or from the lighting up of some tuberculous process. Measles unquestionably renders the tissues very susceptible to tuberculous bacilli; so that infection may result from slight exposure. Acute miliary tuberculosis may follow measles at once, the temperature range being continuous from the outset of the primary disease to death from the complication. General tuberculosis with grave pulmonary involvement may follow so close upon measles as to leave no appreciable interval between. It is sometimes the cause of a secondary fever, which develops soon after the subsidence of the primary fever. Tuberculous disease of the bones and joints subsequent to measles is usually of late occurrence.

*Conjunctivitis.*—Catarrhal inflammation of the conjunctiva is a frequent sequel in most cases of measles. If a child is kept quiet and the eyes protected from the light it usually subsides without special treatment. In some cases, however, it persists even when these precautions are taken. Among poorly fed and ill-conditioned children chronic conjunctivitis is a common sequel of measles. In poorly nourished and anemic children keratitis and corneal ulceration are of not infrequent occurrence. Iritis is also a possible sequel, but is not common. A tendency to granulation of the lids upon slight provocation is sometimes seen for years after an attack of measles.

*Other Complications.*—Nephritis is rare as a complication or sequel of measles, but febrile albuminuria is not infrequent in patients with high temperature. Nervous symptoms occasionally occur, but convulsions at the outset are rare. Acute mania has been reported, but is usually temporary and recovery is complete. Paralysis, though it sometimes occurs, is by no means common. Meningitis also occurs rarely as a sequel. It more commonly follows otitis. Moderate cerebritis or adenitis often occurs and sometimes persists for months, but cellulitis and suppurative adenitis are of rare occurrence. There is a special tendency to tuberculous involvement of the lymph nodes. Endocarditis, pericarditis, and even myocarditis have been reported a few times in the literature. The skin, although the seat of an extensive eruption, is rarely



PLATE XVIII.



Patient with Measles Exhibiting Eruption and Catarrhal  
Inflammation of the Eyes. (Welch and Schamberg.)



injured seriously or permanently. Furunculosis and pemphigus have been known to follow measles, but are unusual. The mucous membranes more commonly become seriously involved than does the skin, and catarrhal inflammations are common. Catarrhal stomatitis is almost as common as conjunctivitis and bronchitis, and ulcerative stomatitis is not infrequent. Gangrenous stomatitis has also been known to occur. Both the latter conditions are seen chiefly in hospitals and tenement houses and are of rare occurrence in well-to-do private practice. Hemorrhages from the mucous membranes are fortunately rare, but not unknown.

*Other Infectious Diseases.*—The occurrence of measles simultaneously with other infectious diseases is not very infrequent. There seems to be a particular tendency to the simultaneous occurrence of measles and pertussis. This is especially common in hospitals. In a recent epidemic in Randall's Island Hospitals this combination of measles and pertussis occurred in many instances and seriously complicated the question of prevention and isolation of each disease. Many cases are recorded of the coexistence of measles and scarlet fever, measles and chickenpox, measles and typhoid fever, and measles and erysipelas. The close association of measles and tuberculosis has already been dwelt upon.

*Prognosis.*—The prognosis of measles differs greatly in private and hospital practice. Death from measles in private practice is rare in children over four years of age. Holt asserts that the mortality is from 4 to 6 per cent., but under two years it is often 20 per cent. or more. It is highest between one and two years, but even at this age uncomplicated measles is not a highly fatal disease. Pneumonia is the cause of death in almost 90 per cent. of fatal cases.

A violent onset with a high temperature warrants a guarded prognosis. The same is true when the eruption is excessive in amount and confluent over wide areas. Pronounced general symptoms with a faint eruption is a grave condition. The same is true of a hemorrhagic or "black" eruption, but it is not as necessarily fatal as is commonly supposed. Age is a very important factor in prognosis. According to statistics recently presented by Holt, measles would seem to be the most fatal between one and two years, even more so than in children between six and twelve months. The mortality is still comparatively high between two and three years. After three years the rate rapidly falls and during later childhood is very small. The temperature is another element of importance in prognosis. A case in which the temperature at no time reaches 104° F. is a favorable one. Every half-degree above that point adds to the danger if it is prolonged. When the temperature continues for any considerable time above 105° F. the prognosis is bad. In other words, measles does not naturally have as high a fever as does scarlet fever, and ranges of temperature decidedly above the average are especially serious. The character of the eruption is still another element to be considered in prognosis. Any considerable departure from the ordinary type is unfavorable. This is particularly true when the erup-

tion is excessively marked or hemorrhagic, and equally so, upon the other hand, when it is faint or ill-defined, with marked constitutional symptoms. A sudden recession of the eruption is also a grave symptom.

Measles has a marked tendency to leave behind it results of a serious nature. Treatment should not be directed solely to saving the life of the child, nor should the prognosis be made up solely with reference to that event. The tendency to tuberculous invasion should never be forgotten, and when the fever persists after the tenth day, even if it is not high, the prognosis should be guarded. The list of chronic affections left in the wake of measles is a long one; bronchitis, pharyngitis, rhinitis, adenoid growths, enlarged tonsils and mesenteric lymph nodes are among the number which should receive consideration. When the fever persists after an attack of measles and the child fails to make satisfactory recovery, search should be made for the various conditions mentioned. As a rule, it will be found that the complicating disease, when obscure, is of a pulmonary or tuberculous nature.

**Prophylaxis.**—The prevailing belief among the laity, too often shared by medical men, that measles is a mild and unimportant disease, leads to great laxness in prophylaxis. In New York City, during the months of March and April, 1904, there were 311 deaths reported from measles. This probably does not include the whole number, for many deaths due primarily to measles were undoubtedly reported as due to pneumonia or some other complication. Any disease which can present a record like this should not be treated as unimportant. It is an unpardonable wrong to unnecessarily expose the children of one's neighbors. Far more care in prevention should be taken than is now often exercised. The advisability of taking particular precaution against the exposure of infants is suggested by the high mortality of measles among children under three years. Delicate children of the so-called scrofulous type and those with hereditary tendency to tuberculosis should be especially guarded against exposure. Early and absolute isolation of the sick is imperative. Isolation of the patient should not be less than twenty-one days and as much longer as purulent discharges may continue. The period of quarantine after exposure should not be less than fifteen days, and twenty days is preferable. Children who have been exposed should be isolated from other children for that period.

The sick-room is likely to prove less dangerous than is the scarlet fever sick-room. Thorough cleansing and ventilation for two weeks is all that is necessary to ensure safety. The infection of measles is not persistent, nor is intermediate infection common; so that prolonged precautions are not necessary. The prevention of the infectious diseases is considered in greater detail under Scarlet Fever (p. 508). Except on certain details which have already been mentioned, the prevention of measles involves much the same precaution as does that of scarlet fever.

**Treatment.**—Measles, like other eruptive fevers must pass through certain definite stages. Notwithstanding claims that are constantly being made in the medical press, no abortive treatment has as yet



been discovered. Ichthyol ointment and other local measures, as well as the use of certain drugs which have been vaunted from time to time as aborting or curing measles, have all been found wanting upon extended trial. The treatment must be symptomatic, and such treatment when judiciously advised and carried out may result not only in the saving of life, but in the prevention of many serious sequelæ.

A room as large and well ventilated as possible should be selected for the measles patient. It should be kept dark and no direct light should be allowed to fall upon the eyes. Full light should not be permitted until the conjunctivæ have assumed their normal appearance. Itching of the lids should be relieved by cold cloths or by the application of cold cream or some bland oil. The eyes should be kept clean by a frequent application of boric acid solution. The same solution or one of normal salt may be used for the nose.

One of the most troublesome symptoms of measles is the hard, metallic cough. It frequently disturbs the patient seriously and breaks his rest. Very little relief, however, can be afforded before the fever begins to subside. It cannot be loosened by the administration of nauseating expectorants. They tend to render the child more irritable and to increase the anorexia, and have but slight effect on the cough. Small doses of opium and codeine aid in allaying the cough, and are quite permissible. Brown mixture (Mist. Glycyrrhiz. Co., U. S. P.) in the form of tablet triturates is as effective as any treatment and is easy of administration. In some cases bromide of sodium acts well in relieving the restlessness and, in a measure, allaying the cough. It may be given in 0.3 gm. (5 gr.) doses every four hours for a child of five years. It should be given in water alone and not in a syrupy mixture. It thus does not disturb the stomach and the child does not object to the slight salty taste. Chloral in doses of 0.2 to 0.3 gm. (3 to 5 gr.) at five years may occasionally be given to relieve restlessness. The cough may sometimes be modified by the use of cool water or cracked ice. As a rule, however, the objection of the patient to being disturbed renders treatment of this nature of little avail.

The fever of measles rarely requires attention. Only when it ranges exceedingly high and affects the patient seriously is it wise to intervene. The effect of the fever upon the patient is a better guide for treatment than is the thermometer. If the child becomes restless or delirious, small doses of phenacetin are admissible. Only enough should be given to lower the temperature moderately and allay the restlessness. Cold sponging is the best treatment for high temperature and is far preferable to the administration of large doses of antipyretics. Water to drink should be given freely if the stomach is not disturbed. Cold bathing based on the Brand method of treating typhoid fever (a bath of 65° F. for fifteen minutes every three hours as long as the temperature remains at 103° F.) has been used in measles, especially by German practitioners. This is wholly unnecessary in most cases, and the injudicious use of baths may do great harm. Cases are very rare in which anything more radical than sponging is required, and sponging is not

often necessary. In the case of hyperpyrexia a bath is admissible. In the case of subnormal temperature a hot bath may be given accompanied by energetic friction of the surface. Stimulants are seldom required in measles. They are indicated in malignant cases and in the presence of complications. The various complications, such as bronchopneumonia and otitis, require the same treatment they would receive under other conditions. A consideration of the details of this treatment is not necessary in this place.

The practice so long in vogue, and still too frequently seen, of sweating the patient and administering hot drinks to bring out the rash, is strongly to be discouraged. In the great proportion of cases the eruption will come out in due time and nothing is to be gained by rendering the patient wretched and uncomfortable. In the case eruption is really retarded or is faint, the patient may be wrapped in a sheet wrung out of hot water, but this is rarely necessary. The use of the iodides, acetate of potash, and Dover's powder is rarely productive of good. In fact, the more simple the treatment of measles is made the better are the results. Uncomplicated cases of average or even of severe type require very little medication. Treatment directed toward the bronchial catarrh is often all that is required. Active cathartics should be avoided as far as possible, for their use is not infrequently followed by diarrhea. If constipation is present, it is best to relieve it by enemata; but if the tongue is heavily coated small doses of calomel and soda may be given with good effect. If the enemata are not productive of a result, a mild saline cathartic, such as citrate of magnesia, may be administered.

The eyes should receive more careful attention than is frequently given to them. The room should be kept well darkened, and even after the light is admitted the use of the eyes should be much restricted, and, as stated, boric acid solution should be used to wash the lids. The acute inflammation to which they are subjected, as well as the debilitating influence of the disease, renders the eyes themselves, as well as the ocular muscles, particularly weak and sensitive. In some cases their use should be restricted for several weeks after recovery. The child should not be permitted to go to school until the eyes are in a strictly normal condition. Phlyctenular conjunctivitis with its army of dangerous complications, including ulceration of the cornea, is often witnessed in the dispensaries as a sequel of measles. Most text-books do not lay sufficient stress on the importance of keeping the lids aseptic by careful cleansing and on not using the eyes too soon.

During convalescence unusual care should be exercised in avoiding unnecessary exposure. The various sequelae should receive proper attention and the particular susceptibility to tuberculosis should not be forgotten. If the child continues anemic or the cough persists, cod-liver oil and iron are particularly indicated. In such cases a change of climate will often accomplish more than medicine. If tuberculosis is to be feared, either from hereditary predisposition or other cause, the patient should not be dismissed permanently, but should be kept under occasional observation. As tuberculous symptoms sometimes develop

at a considerable interval after the immediate effects of the disease have disappeared, the necessity for good food, cod-liver oil, and an open-air life in suitable weather should not be forgotten.

### RUBELLA.

By FLOYD M. CRANDALL, M.D.

Rubella, German Measles, or R  theln, is an acute, infectious, and contagious disease, presenting somewhat varied symptoms. It is an entity and not a modified form of the other eruptive diseases. Typical cases present the following features: After an incubation of about fourteen days a rash appears on the face and extends rapidly over the body, reaching its height within twenty-four hours, and usually disappearing by the end of the third day. There is sometimes a short, indefinite stage of invasion; the temperature is not usually over 101   F., and rarely continues over three days; a slight desquamation sometimes occurs; complete recovery without complication is the rule. One of the most characteristic symptoms of rubella is enlargement of the post-cervical lymph nodes.

**Etiology.**—Nothing is known of the bacteriology of rubella. It is rarely seen in children under six months, but above that age the predisposition seems to be universal, its occurrence not being modified by sex or age. It occurs usually in epidemics, which are most frequent during the winter or spring. It is less contagious than measles. Ashby and Wright assert that susceptibility seems to vary strangely at different times and in different places, so that in some epidemics it seems to be very contagious and in others slightly so. This probably accounts for the varying opinions regarding its contagiousness expressed by different authorities.

The term *German measles* is an unfortunate one, for it leads to much misunderstanding, particularly among the laity. Rubella does not protect against measles and scarlet fever, and, on the other hand, these diseases do not protect against rubella. A marked demonstration of this recently came under my own observation. On Tuesday the three little sons of a well-known surgeon awoke covered with a profuse eruption of rubella. The eruption was at its height on Wednesday morning, had faded on Thursday, and disappeared on Friday. On Saturday afternoon the second boy was taken ill (see Fig. 115) and passed through a typical attack of measles. This boy I had attended two years before through a typical case of scarlet fever lasting six weeks. The two other boys came down with measles on the twelfth day after the Saturday upon which the first one became ill.

**Period of Incubation.**—The extremes are from six to eighteen days, possibly twenty-two days. The average is probably fourteen days. The period varies considerably in cases occurring in the same epidemic.

**Period of Contagiousness.**—Rubella may be contagious for a few days before the rash appears and continue so until complete recovery,

a period sometimes of two weeks. It is most contagious on the three days following the appearance of the rash.

**Clinical Types.**—As there is grave doubt as to the existence of the "fourth disease," it still seems best to describe rubella as occurring under two types—the measles type and the scarlatinal type.

*The Measles Type.*—After a stage of invasion lasting but a few hours and marked by malaise and, perhaps, feverishness, a rash appears on the face and neck and spreads rapidly over the body. This stage of invasion is frequently lacking, the rash being the first evidence of illness. Sometimes the child wakes in the morning covered with the rash. The individual lesions are of a size and appearance to be very suggestive of measles. As a rule, the spots are of a pale rose-red color, larger than those of scarlet fever, but smaller and less blotchy than those of measles. They are rarely grouped and the skin does not assume the scarlet hue. The rash is most intense on the second day, but rapidly fades and is often not discernible after the third day. The fever is the highest on the second day, but in many cases the child is but slightly ill at any time. In others there is considerable malaise and heaviness on the first three days. Occasionally there is fever, nausea, headache, and all the evidences of acute illness. Desquamation, when evident at all, appears soon after the eruption has subsided. A faint pigment sometimes appears for a few days after the rash has gone, but does not persist as does the staining following measles. The throat is often dry and red, but exudates are exceedingly rare. Recovery is prompt and there are rarely any complications or sequelae.

*Scarlatinal Type.*—In this type the constitutional symptoms are similar to those of the measles type. The two types differ chiefly in the appearance of the rash. The eruption is copious and very similar to that of scarlet fever. It is usually, however, less punctate than that of scarlet fever and more of a rose tint. There is a uniform redness of the skin, but the little points about the hair follicles are faint or entirely absent. In sporadic cases it is often impossible to make a diagnosis from the rash alone, and sometimes it is necessary to wait for desquamation to settle the question. Even in the distinctly scarlatinal type small areas will occasionally be found, especially on the forehead and arms, in which a maculopapular eruption appears. In cases of doubt the whole body should be examined, for areas of the measles type of eruption may be found which will aid in making a diagnosis. The scarlatinal type is less common than the measles type.

Rubella sometimes appears in a more severe form than that here described. The prodromal stage is decided, the eruption is marked, the temperature ranges as high as 103° F. and continues for three or four days, and the child seems decidedly ill. Vomiting, headache, and delirium may be present, and the diagnosis may be difficult.

**Symptomatology.**—*The invasion* in very rare instances is marked by a convulsion, chill, or severe headache. Usually if there is any stage of invasion the symptoms are those common to mild febrile conditions.



*The fever* is rarely high. The temperature is often not over 100° F. but in more severe cases may reach 102° or even 103° F. It is highest on the second day and often lasts but one day. It is impossible to present a chart which can be regarded as typical. The pulse and respiration present nothing characteristic.

*The throat* is usually red and the eruption may sometimes be seen on the roof of the mouth. Exudates are not seen except occasionally as a complication. Forchheimer describes an "enanthem" which he believes to be characteristic of rubella. It is seen on the first day on the uvula, but not on the hard palate, and consists of bright rose-red points of minute size.

*The eruption* may cover the entire body or may be limited to small areas. It is rarely absent from the face. In rubella and measles the rash usually appears on the lips, but in scarlet fever the region about the mouth usually remains free. The eruption is rarely confluent except on the face, and is seldom, if ever, hemorrhagic. It is sometimes so elevated as to have a shotty feel as the finger is passed over the skin. Itching is very common on the first day. Authors differ widely in their statements as to the duration of the eruption. In a recent epidemic in New York it was not unusual to see a profuse eruption disappear entirely at the end of forty-eight hours, but commonly some evidence of the eruption could be found for three or four days.

*Desquamation* usually occurs slightly, but in some cases cannot be detected. It is light and branny and rarely, if ever, profuse. A doubtful case followed by marked desquamation may safely be regarded as scarlet fever and not rubella.

*Swelling of the lymph nodes* is one of the most constant and distinctive symptoms of rubella. So constant is its occurrence that the diagnosis should be made with caution when it is not present. The lymph nodes most frequently involved are the cervical, the postcervical, and the suboccipital. A nest of small lymph nodes found low in the neck behind the sternomastoid muscle is especially characteristic of the disease. In the case of the three boys referred to on the previous page, the involvement of the lymph nodes during the rubella was excessive. It rapidly subsided and during the measles no nodes could be felt. Although rubella is an extremely mild disease, the peculiar enlargements of the lymph nodes, the marked eruption, and its close simulation of more serious diseases render it of considerable interest.

**Diagnosis.**—The differential diagnosis between rubella, measles, and scarlet fever is often very difficult and sometimes impossible. It is, however, very important. It is unfortunate to isolate a child for five or six weeks who is simply suffering from rubella, but still more unfortunate to allow a mild case of scarlet fever to go at large through making the opposite mistake. As rubella usually occurs in epidemics, it is the part of wisdom to regard every suspicious sporadic case as mild scarlet fever or measles until the diagnosis can be made with certainty. Certain drug rashes, especially that of belladonna, closely simulate the rash of rubella, and there are many unclassified eruptions which may easily

be mistaken for it. In every doubtful case the possibility of a drug eruption should be investigated as well as the condition of the digestive tract. This subject is further considered under the diagnosis of Scarlet Fever (p. 505).

**Prognosis.**—The prognosis of rubella is invariably good. There are, in fact, but few eruptive diseases so little liable to complication or serious symptoms.

**Treatment.**—Most cases require no treatment other than quiet in bed while the eruption lasts, a liquid diet, sponging, and anointing with vaselin or cold cream. In the more severe cases the treatment is that required by all febrile conditions. If the case is severe enough to require definite treatment, the measures advised for measles may be appropriately adopted.

Isolation for two weeks at least is necessary to prevent the spread of the disease, and the prophylactic measures advised for measles should be carried out.

#### FOURTH DISEASE.

By FLOYD M. CRANDALL, M.D.

In 1900, Clement Dukes, physician to the school at Rugby, published a description of what he believed to be a disease not before described, to which he tentatively gave the name of "Fourth Disease." As described by him the only difference between rubella and the fourth disease is in the rash. In fact, the disease he described is virtually that which I have described as the scarlatinal form of rubella. It is quite true that the two forms of rubella seem like different diseases, but not more so than do different types of scarlet fever. The extended chart of differential diagnosis given by Dukes describes identically the same disease except as to the rash, and he even admits that in the same patient the eruption sometimes resembles measles and later scarlet fever. It is rare to find a case of the scarlatinal type of eruption in which areas of the measles type cannot also be found upon careful search. In other cases the eruption is mixed, and if we grant the existence of the fourth disease can only explain such a case as suffering from both diseases.

The question has been considerably discussed during the past four years. A very careful study of 32 cases of rubella was made by Watson Williams, many of the cases being what Dukes would call the fourth disease. He is inclined to question the existence of the fourth disease, believing that the cases thus described are either rubella or mild scarlet fever. Pleasants, of Baltimore, also had opportunity to especially study such cases, and concludes that the existence of a new exanthematous disease has not been established. After an extended review of the four diseases in the *Practitioner* for February, 1902, Ker concludes that the fourth disease is either mild scarlet fever or atypical rubella. From a study of the literature and considerable experience with the

three diseases, it seems to me that we have not sufficient evidence to warrant us in describing a fourth. More proof is needed before we can accept it as a clinical entity.

### ERYTHEMA INFECTIONOSUM.

By JOHN RUHRÄH, M.D.

Erythema Infectiosum is a slightly contagious disease of childhood characterized by a maculopapular, reddish rash, and by slight or no subjective symptoms. This condition was described as a separate disease by Escherich in 1896. It has not as yet (January, 1905) been observed in America.

**Etiology.**—The disease occurs in epidemics, most frequently in spring and summer. It usually affects children between the ages of four and twelve. Infants under one year are apparently immune. It is but feebly contagious and close contact would seem to be necessary to communicate the disease. An attack of this disease does not protect from measles, scarlet fever, or German measles, and *vice versa*. No organism has as yet been described in connection with this erythema. The incubation period is given as being from six to fourteen days.

**Symptomatology.**—Prodromes are rare. The rash is usually the first thing noted. It appears first on the face, covering the cheeks with a uniform, rose-red flush, which is slightly raised above the surface and has rather abrupt borders. The lips are free and the forehead and chin but slightly spotted with small patches. This is hot to the touch, but is not sensitive and does not itch. It disappears on pressure, but returns immediately. The rash appears next on the extremities and trunk and it spreads from above downward. On the trunk there are more or less discrete, crescentic patches, varying in size from one-eighth to half an inch. The rash is marked on the buttocks and the extensor surfaces of the arms and legs. In these latter locations it varies in color from rose-red to a brownish-red, and it runs together, forming gyri and networks of a map-like character. The eruption fades from the face in from four to five days and a little later from the body. In all the rash is present from six to ten days. There is no subsequent desquamation or pigmentation. In some cases it disappears and later reappears. The mucous membranes are not affected.

Other symptoms are rarely present. In a few cases there has been slight fever for a day or two. Occasionally other things have been noted, as sore throat, slight reddening of the conjunctivæ, and rarely joint pains. There is no enlargement of the lymph nodes.

**Diagnosis.**—This depends largely on the recognition of the rash. Scarlet fever has high fever, marked constitutional symptoms, and a more or less uniform rash. In measles the fever, constitutional symptoms, the catarrhal symptoms, and Koplik spots are sufficient to distinguish it. In rubella the presence of enlarged nodes and the punctate

eruption on the soft palate are points of difference. Urticaria is easily diagnosed by the itching, and drug rashes, from the history of having taken drugs. Erythema exudativum multiforme begins on the hands and feet, becomes vesicular, has marked constitutional symptoms, and lasts much longer.

It should not be confused with what Dukes<sup>1</sup> has described as "Fourth Disease," in which the scarlatiniform eruption is said to appear suddenly over the body. (See article on Fourth Disease, p. 538.)

**Prognosis.**—This is favorable. There are neither complications nor sequelæ.

**Treatment.**—This is symptomatic.

<sup>1</sup> London *Lancet*, July 14, 1900.



## CHAPTER XXI.

### VARICELLA—VACCINIA—SMALLPOX.

#### VARICELLA.

By FLOYD M. CRANDALL, M.D

**VARICELLA**, or Chickenpox, is an acute, infectious, and contagious disease occurring almost exclusively among children. In typical cases, after an incubation of fourteen days, a vesicular eruption appears and continues to develop in crops for three or four days. Each vesicle dries, forms a crust, and falls off, usually leaving no pit or mark. While the eruption is appearing there may be mild febrile symptoms, but the disease is rarely serious. The term varicella (diminutive of variola) was given at a time when the disease was not fully differentiated from smallpox.

**Etiology.**—Varicella is unquestionably an infectious disease, but the micro-organism has not yet been discovered. It must reside in the vesicles, for the disease may be transmitted by inoculation of the vesicle serum. It may also be transmitted by direct contact, it being, in fact, almost as contagious as measles. Intermediate infection through a third person is also possible. The dried crusts contain the infective agent, and may be the means of transmitting varicella as the desquamation scales transmit scarlet fever. Baader, of Bâle, found in 584 cases that 98 per cent. of the patients were under ten years of age and 65 per cent. were under five years. Sex and season have no influence on its occurrence. Epidemics, while common, are not usually very widespread. The infective principle may remain active for many weeks.

**Period of Incubation.**—This is rarely, if ever, less than twelve days or more than sixteen. The most common period is fourteen days.

**Period of Contagiousness.**—Varicella is contagious from the outset until the last crust has fallen and the purulent discharges have ceased, a period not usually less than fourteen days. It may be longer than this.

**Clinical History.**—The eruption is usually the first symptom noticed. Occasionally there is a period of invasion lasting for twelve or even twenty-four hours, marked by lassitude, feverishness, and, perhaps, pains in the head and back. The eruption is vesicular, but the lesions begin as small, red papules. The papular stage, however, is very short. Usually at the physician's first visit a number of vesicles are already well developed, but rose-red papules are also present. The first lesions appear upon the face and trunk, especially upon the back, where their development is usually most typical (Figs. 117 and 118). In this early stage the vesicles consist of little round blisters filled with clear

fluid, surrounded by a small zone of redness. The skin between the lesions is normal. Most of the vesicles are unilocular and collapse when they are pricked. They are rarely, if ever, confluent. The clear serum of the vesicle becomes cloudy and within twenty-four or thirty-six hours begins to dry so that a scab is formed. The vesicles appear in crops even in the same locality. Hence, papules, new vesicles, old vesicles, and scabs may be found in the same patient a few days after the onset. The attack is usually at its height on the third or fourth day and the acute symptoms are passed within a week or ten days, but the scabs frequently do not all fall before the end of the third week and sometimes later.

FIG. 117



Chickenpox.

**Symptomatology.** *Temperature.* The fever of varicella is extremely variable. In mild cases there is often none whatever. Usually there is slight elevation of temperature for one or two days and not infrequently, in the more severe cases, the fever continues for four or five days or even longer. It is usually intermittent in type and may range between 99° and 102° or 103° F. In the more severe but rare cases it may reach 104° F. It seems to me impossible to present a chart that could be called typical of varicella.

*Eruption.*—The lesions of the typical eruption have been well described as looking as if drops of hot water had fallen upon the skin and raised

small, round blisters, with a narrow, inflamed zone around each. When the skin is thick, as on the palms and soles, there is no red zone, the vesicle lying alone in the normal skin. The contents of the vesicles is at first clear and soon becomes cloudy, but not purulent unless they are irritated or infected. The number of vesicles ranges from a dozen or a score to many hundred and are most profuse and typical on the back and shoulders. But few appear upon the face; in some cases none are seen there. The vesicles begin to dry in the centre and frequently present an umbilicated appearance, when the process is partially completed. Scabs or crusts are soon formed, which fall in from seven to twenty-one days according to the depth to which the process extended.

FIG. 118



\*Chickenpox vesicles surrounded by reddish areole. (Welch and Schamberg.)

Pitting is rare. This sometimes happens, however, when the vesicle has involved the true skin. It is most common on the face. Deep ulcerations which may last for several weeks sometimes occur. They are most common in anemic, poorly nourished children, especially those of tuberculous tendency.

**Complications.**—Varicella is very rarely complicated, although two grave complications are possible. It is a strange fact that in a disease so mild, complications, when they do occur, should be so serious. These complications are gangrenous dermatitis and erysipelas.

**Gangrenous Dermatitis.**—The so-called *varicella gangrenosa* is simply gangrenous dermatitis taking its origin from varicella lesions. It is most common on the neck, chest, and upper part of the trunk. It is

but little amenable to treatment and runs its course in from seven to twenty days and usually terminates fatally. It probably never occurs in perfectly healthy children, but is usually seen in tuberculous and ill-nourished patients of hospitals and dispensaries.

*Erysipelas.*—While not frequent in occurrence, erysipelas is probably the most common complication of varicella. Nephritis has occasionally been reported as a complication. Adenitis of marked type may also occur. Other complications reported from time to time are probably not more than coincidences. Varicella, like all the eruptive fevers, may occur in conjunction with one of the exanthemata. The combination of varicella and scarlet fever is probably the most common. Relapse and recurrence are extremely rare.

*Diagnosis.*—The only disease with which varicella is likely to be confounded is smallpox of mild type. Smallpox begins with a stage of invasion marked by fever, backache, headache, and drowsiness and often vomiting and delirium; varicella begins with an eruption preceded by a very short and mild stage of invasion and often by none whatever. In smallpox the lesions begin as papules and remain so for one or two days, when they develop into vesicles and finally by the eighth day into pustules; in varicella the papules change into vesicles in a few hours, become cloudy and dry into crusts before it is time for the variola vesicle to become a pustule. In smallpox the lesions are all of the same age; in varicella, papules, vesicles, and crusts are all present in the same locality at the same time. In smallpox the lesions are multilocular and truly umbilicated; in varicella they are mostly unilocular and appear umbilicated only as they begin to dry in the centre.

*Prognosis.*—The prognosis is always good in patients in ordinary health. Only in the marasmic, ill-nourished, and tuberculous are untoward symptoms to be expected.

*Treatment.*—The varicella patient is capable of transmitting the disease while the crusts remain. It is wrong to permit the exposure of infants and weakly children. It is also wrong to expose the children of other people even to a trivial disease, for one can never know what inconvenience it may produce. Hence, there are many cases in which isolation should be enforced from the first symptoms until the crusts have fallen. Medicinal treatment is rarely required. In the more serious cases symptoms should be treated as they arise. Itching or irritation of the skin may be relieved somewhat by a carbolic wash or camphorated vaselin. Lesions which become irritated or broken should be dressed with a boric acid or other mild ointment and managed with antiseptic care.



## VACCINIA.

Vaccinia, or Cowpox, is an acute, infectious disease of the cow characterized by a vesicular eruption upon the udder and teats. The disease may be communicated to man by inoculation of the lymph from these vesicles and affords protection, for a variable period, against smallpox.

Whether smallpox and cowpox are the same disease or are separate entities is still a subject of discussion. The weight of evidence, Osler believes, favors the view that cowpox is variola modified by transmission.

The first vaccination on a human subject was performed by Edward Jenner on May 14, 1796. It was a matter of popular though local observation that persons who had been inoculated by cowpox rarely contracted smallpox. Little James Phipps was the first subject. His vaccination ran a typical course and after several subsequent unsuccessful trials the boy was taken through a smallpox hospital without the slightest harm. Two years later Jenner published his observations in a little book of seventy-five pages, entitled *An Inquiry into the Cause and Effects of the Variolæ Vaccinæ, a Disease Discovered in Some of the Western Counties of England, Particularly Gloucestershire, and Known by the Name of Cowpox*. From the publication of this little book the adoption of vaccination was very rapid. The first vaccination in America was performed in Boston on July 8, 1800, by Dr. Benjamin Waterhouse, Professor of Physic at Harvard. The operation was introduced into the Southern States through the personal efforts of Thomas Jefferson, then President, who fully understood the ravages of the disease among the black population.

**Protective Power of Vaccination.**—In determining this question certain historical and statistical study is necessary. We must know what smallpox was before vaccination and what it has been since. In a paper entitled "A Century of Vaccination," published in *American Medicine*, December 7, 1901, I considered the subject from many standpoints and summarized a large quantity of statistics. Space here permits reference to but a few facts.

A hundred years ago smallpox was justly regarded as "the attila of diseases, the very scourge of God, overrunning countries and destroying populations." When Jenner performed his first vaccination, it was causing one-tenth of all the deaths of the human race. Bernouilli, the mathematician, estimated that more than 60,000,000 of the inhabitants of Europe died of smallpox during the eighteenth century. Others place the number even higher. Specific proof of its fatality is shown by Cowan's vital statistics of Glasgow. In that city between 1783 and 1792, 36 per cent. of all deaths under ten years were due to smallpox. One-third of all the deaths in Europe under ten years were due to the same cause. When smallpox was introduced into Mexico by the Spaniards in 1520, 3,500,000 died within a few years. In 1737, in

Iceland, 18,000 in a population of 50,000 died in a single year. It is believed that 6,000,000 North American Indians fell victims to its ravages.

One hundred years ago smallpox was the most widespread disease which affected the human race. To-day many physicians of large experience have never seen a case. Some marvellous power has been at work to produce this change. Isolation and improved sanitation are valuable aids in suppressing the disease, but they cannot explain these changed conditions. In Sweden the death rate from smallpox for twenty-five years preceding vaccination per million living was 2045. Under optional vaccination it fell to 408, under compulsory vaccination to 155, and for ten years under more rigid laws to 5. In Germany after the rigid law of 1874 the rate per million fell from 309 to 15, and for ten years has averaged 7. In the German army, in which the vaccination law is most thoroughly enforced, there has been but one death from smallpox since 1874. The following figures are given on the authority of the *Practitioner* and of Sir George Buchanan for the Sheffield epidemic of 1887-88:

Attack rate per 1000 in the non-vaccinated . . . . .	94; death rate, 51.00
" " " " " once vaccinated . . . . .	19 " 1.00
" " " " " revaccinated . . . . .	2 " 0.09
1 in 1300 of the vaccinated died; 1 in 20 of the unvaccinated.	

**Clinical History.**—The clinical history of normal vaccination is fairly uniform. Any marked deviation from the normal course may vitiate the value of the result. It is entirely reasonable to insist that in a procedure like vaccination certain requirements should be fulfilled. A vaccination sore should pass through certain well-defined stages. If it does not do so it is not an adequate vaccination, and cannot be expected to confer full protection.

**Incubation.** There is usually some slight irritation after vaccination which subsides completely and nothing may be apparent for two or three days.

**Eruption.**—On the third or fourth day after vaccination a faint redness appears at the point of inoculation. This redness gradually increases and a little reddish papule is formed. The papule gradually changes into a vesicle which on the fifth or sixth day contains a thin, transparent fluid. By the eighth day the fluid has become yellowish in color and in the centre a little depression may be seen. About this time a circle of inflammation, the areola, appears about the vesicle. By the tenth day the inflamed skin is tense and painful and the vesicle has become a pustule. By the twelfth day the vesicle begins to dry, and by the fifteenth day a crust has formed (Figs. 119, 120 and 121). This crust is of mahogany color, rough, but thinner in the centre than at the edges. It rarely falls before the end of the third week. The scar is at first red, but soon fades, and has a pitted or streaked appearance.

The following series of lesions is necessary for satisfactory vaccination—papule, vesicle, pustule, scab, and scar.

*Constitutional Symptoms.*—In most cases there is a period of feverishness, fretfulness, and malaise. This usually begins on the fourth or fifth day, but may begin as early as the third or as late as the eighth

FIG. 119



Infant born of a varicelous mother in the Municipal Hospital, Philadelphia; vaccinated on day of birth; protection complete; photographed on ninth day. (Welch and Schamberg.)

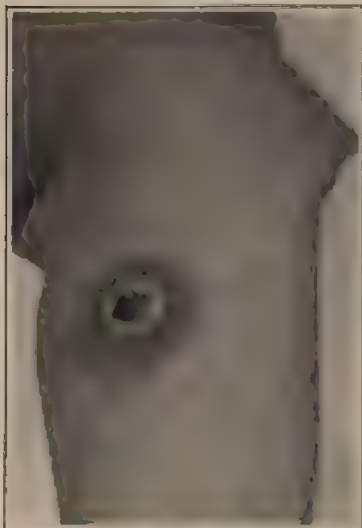
FIG. 120



Infection in an adult, showing vesicles upon the eighth day. (Welch and Schamberg.)

or tenth day. The temperature usually rises to  $100^{\circ}$  or  $101^{\circ}$  F. the condition continues for three or four days. The lymph nodes, axilla or groin are sometimes slightly enlarged and may be painful. In other cases the symptoms are more decided. The fever is sometimes reaching  $103^{\circ}$  or even  $104^{\circ}$  F., is intermittent in type and continues for four or five days. In rare cases an even more severe fever is seen. In babies the fever and general symptoms are often

FIG. 121



Vaccine vesicle on ninth day, showing pronounced areola. (Welch and Schunberg.)

there being simply a day or two of discomfort or loss of appetite. The modern methods of vaccination produce general symptoms as well as a local sore are usually less marked than they formerly were.

**Irregular Vaccination. Local Variations.**—The local sore may vary greatly from that just described. A large and angry sore is sometimes seen, but it confers no great immunity than does one of normal type. On the other extreme a very small sore is not uncommon with glycerine lymph. The size, however, does not invalidate the result, if it passes through the various stages in proper order. The value of a vaccination should be doubted if it progresses abnormally fast, so that a crust is formed at the end of a week. It should also be doubted if the contents of the vesicle is bloody.

If the sore discharges pus. Traumatism after the seventh day may render the vaccination valueless, but it is wise to make a second vaccination. A sore after a vaccination is not in itself a guarantee that immunity has been conferred. It should pass through the five stages in due order. Any marked variation from the normal course casts suspicion upon the adequacy of the vaccination.

A condition known as the "raspberry excrescence" sometimes takes the place of the ordinary vaccination sore. It rises out of the skin and is usually of a dark-red color and of slightly lobulated appearance. It is not sore and does not discharge pus. It usually disappears after two weeks, but occasionally persists for several months. It is supposed to be due to weak virus, or to virus containing some part of a bacterium. It does not confer immunity.

As a rule, the vaccination sore is the only lesion which occurs on the skin, but sometimes secondary pustules appear about the primary sore. Less frequently a generalized eruption occurs and the child may be actually ill. This is sometimes pustular and resembles smallpox. At other times the eruption consists of dusky, mottled patches.



rose-colored eruption which continues for two or three days and is followed by slight desquamation. I once saw a case in which a profuse eruption on the trunk and neck appearing eight days after vaccination consisted of large, oval blotches of deep-red color, surrounded by a lighter areola which shaded off into normal skin. Secondary pustules are not infrequently caused by inoculation by the finger-nails from the primary sore.

*Constitutional Variations.*—A vaccination may be efficient even without the constitutional symptoms. This is not uncommon in infants. The character of the sore, not the general symptoms, should be the guide. On the other hand, severe symptoms may occur without modifying the result.

*Complications.*—The various complications of vaccinia and the time of their occurrence are thus classified by Acland: During the first three days, erythema, urticaria, vesicular and bullous eruptions, invaccinated erysipelas. After the third day and until after the pock reaches maturity, urticaria, lichen urticatus, erythema multiforme, accidental erysipelas. About the end of the first week, generalized vaccinia, impetigo, vaccinal ulceration, glandular abscess, septic infection, gangrene. After the involution of the pocks, invaccinated diseases.

Cellulitis is the most common complication. It is due to bacterial infection and may be the result of infected virus, carelessness in performing the operation, or to later infection. In mild cases there is simply more intense inflammation in the areola than is normal. In a more severe type pus forms under the scab and about the sore and the areola is of unusual size. In still more severe cases an excavated ulcer may be found, which is extremely stubborn and difficult to heal. These sores sometimes last two months. More or less enlargement of the adjacent lymph nodes is likely to follow and suppuration may occur in them, but this is very rare. While cellulitis due to infection by pyogenic bacteria is common, erysipelas is not often seen. It is a very possible complication, however.

Most of the arguments against vaccination date back to the time of Birch and Rogers, in 1805, and are based to a considerable extent upon an experience when arm-to-arm vaccination was practised. By that method blood diseases were occasionally transmitted. The bovine species from which all vaccine lymph used in this country is now obtained is not susceptible to syphilis and that disease is never transmitted by vaccination with bovine lymph. Syphilis is a peculiar disease in its manifestations. Infants affected with syphilis are very frequently born apparently healthy, and the first signs usually show themselves during the third or fourth weeks. Many cautious physicians, therefore, refuse to vaccinate a child before the end of the sixth week. The disease has many times been charged to vaccination and physicians have received undeserved censure, when it was in fact the disease was inherited. Tuberculosis is not transmissible by modern vaccine lymph. Acland thinks it doubtful whether it has ever been so transmitted. It is extremely doubtful whether the tubercle bacilli ever appear in the lymph even in animals

suffering from the disease. To guard against any such chance, however, the leading makers examine postmortem every calf from which lymph has been taken. If any evidence is found of tuberculosis or any other disease, the lymph from that animal is rejected. A few great firms make much of the lymph now used in this country, and could not afford to have accidents happen from the use of their products. It is unquestionably true that tetanus has been conveyed by vaccine lymph. In 1901 several cases occurred in the United States and two-thirds of them were traced to lymph procured from one source. This experience suggests the importance of government supervision over the production of vaccine lymph. There is no authentic case on record in which cancer has resulted from vaccination. The tendency of certain diseases to be dormant and appear at certain times or to be waked into activity by slight exciting causes is to be considered in studying the supposed complication of vaccination. This is notably true of tuberculosis, syphilis, and eczema.

**Technique.**—The outer aspect of the left arm at the insertion of the deltoid and the outer aspect of the left leg, one-third of the way from the knee to the hip, are the points usually selected for vaccination. A site over a bone like the shin or a spot over a tendon should never be selected. In young children the leg is most readily reached and can be most easily cared for. In older children the arm can be most easily protected from dirt. It should be selected for children who will not be closely cared for.

Vaccination is a surgical operation and should be done with surgical cleanliness and care. The skin should be washed with warm soap and water or with alcohol. Other antiseptics should not be used, for if they are not thoroughly removed they may harden the skin and neutralize the vaccine virus. The best instrument is a common cambric needle and a fresh one should be used for each patient. The needle should be sterilized by boiling or heating in an alcohol flame just before using. The skin is put slightly on the stretch and with the point of the needle four or five scratches a quarter of an inch long are made. They should not be deep enough to draw blood, but no harm is done if a few minute points of blood appear. These are crossed by other scratches not made too close together. The virus is then dropped on to this area and well rubbed in with the blunt end of the needle. It is then allowed to dry before it is covered, which often requires twenty minutes. A method of vaccinating much in vogue of late consists in scraping off the surface layers of the skin until a pink, oozing spot is obtained without actual bleeding. The vaccine virus is well rubbed in and allowed to dry. I used this method in some 200 cases, but found that I secured more certain results by the older method of scarifying.

**After Care.**—The wound should be covered with an aseptic bandage and should be kept covered as any other surgical wound would be. Were this principle universally carried out a great source of trouble after vaccination would be eliminated. Serious sores are caused by extraneous germs. Their introduction may result from lack of care in

performing the operation, but more often from improper care or injury after it has been performed. Infection in older children is more common when vaccination is done on the leg, because it is more apt to be infected with dust. Little girls who are vaccinated on the leg and are then allowed to run about with the sore unprotected are particularly liable to develop complications. Such complications may be prevented by a protective dressing. A heavy surgical dressing is not advisable, as it sweats and softens the scab. Shields are more apt to cause trouble than to prevent it. This is particularly true of those that are covered or have hard edges. Talcum powder should be freely used, particularly if the sore is moist. A light gauze bandage which is changed frequently is the best dressing when it does not stick in the sore. When there is considerable soreness or the dressing sticks, a light wire shield or a perforated felt shield of large size is admissible and often gives much comfort. It should be changed or cleansed frequently. A few turns of light gauze bandage should be placed over it.

If the wound becomes infected and purulent it should be cleaned out like any other wound and dressed surgically. Poultices and oily applications should not be used after vaccination. In fact, as long as the wound is pursuing a normal course no application should be made to it. Protection is all that it requires. If it becomes too moist or oozes serum, it may be dusted with bismuth subgallate, aristol, or some simple dusting powder.

*Selection of Lymph.*—Bovine lymph is now used almost wholly in this country and should be employed exclusively. When the operation is properly performed, the danger of conveying disease is completely removed, which is not true of humanized virus. Glycerinated lymph should be selected as the most perfect product yet devised. Saprophytic germs cannot live in glycerin in hermetically sealed tubes. When properly prepared such lymph is sterile and cannot be contaminated in handling, as so often happens with quill and ivory points.

*Time for Vaccination.*—Although young infants bear vaccination well, as a rule, for reasons already referred to, it is unwise to vaccinate during the first or even the second month. In well-nourished healthy infants the third month is the best time for vaccination. They are usually less ill than when they are older, and complications are less liable to occur than during the first weeks. In delicate children it is well to wait until the nutrition is fully established and the general condition assured. It is unwise, unless smallpox is prevalent, to vaccinate when the child is acutely ill or is suffering from any active disease of the skin or lymphatics, particularly eczema or urticaria.

*Revaccination.*—In considering this subject, it is wise to determine first just what is to be expected from vaccination. For this purpose we cannot do better than quote the words of Jenner, whose claims for vaccination, though always positive, were judicious and by no means extravagant. His own words were: "Duly and efficiently performed, it will protect the constitution from subsequent attacks of smallpox as much as that disease itself will. I never expected that it would do more,

and it will not, I believe, do less." It is well known that smallpox is sometimes repeated in the same subject, that is, that immunity is not always lifelong. No competent authority claims that the immunity conferred by vaccination is always lifelong. In many cases it is of limited duration, being sometimes as short as six or seven years. In a few cases one vaccination seems to afford lifelong immunity, and in most cases two successful vaccinations are sufficient.

Experience of more than a century has strengthened and confirmed the teachings of Jenner. Some of the lessons taught by this experience may be summarized as follows: 1. The first lesson cannot be better stated than in the words of the Berlin Board of Health: "Vaccination in infancy, renewed at the end of childhood, renders an individual practically as safe from death from smallpox as if the disease had been survived in childhood and almost as safe from attack." 2. The duration of the immunity conferred by vaccination is variable. In many individuals vaccination in infancy and revaccination in childhood is sufficient for life protection. In a limited number immunity is lost in five or six years.

It is never possible to know with certainty to which class an individual belongs. In the face of an epidemic, therefore, vaccination of all persons who have not been vaccinated within five or six years is giving what the lawyers call the benefit of a reasonable doubt. Every one who has been vaccinated in infancy and childhood should be vaccinated not less than once in adult life. 3. The immunity conferred by vaccination is in direct proportion to the thoroughness with which it is performed, and this is shown with considerable accuracy by the character and number of the resulting scars. 4. Vaccination in infancy alone is not sufficient to prevent smallpox among the adult population.

### SMALLPOX.

Smallpox or Variola is an acute, infectious, and very contagious disease marked by a pustular eruption and a fever which lasts for three or four days and is followed by a secondary or suppurative fever on the eighth or ninth day. It is one of the most virulent of the contagious diseases, and those who are exposed, if unprotected by vaccination, are almost invariably attacked. Smallpox has appeared in nearly every country of the globe and is of very ancient date. The "great plague" described by Galen was probably smallpox. Further facts regarding the disease will be found in the section on Vaccination.

**Etiology.** *Exciting Cause.*—Until very recently it was necessary to write of smallpox as of the other eruptive fevers, that the micro-organism was unknown. It now seems probable that the exciting micro-organism of the disease has been discovered. Councilman has reported the discovery of intracellular and intranuclear bodies in the lesions of smallpox which are probably the specific cause of the disease. These bodies are protozoa, the *cytomyxetes variola* (Guarnièri). In a more recent



communication (*The Journal of Medical Research*, February, 1904) Councilman and a half-dozen collaborators present evidence which seems to confirm the belief that this is in fact the micro-organism of smallpox. His conclusions are summed up in the following words: "In the early stage of the specific lesions of the skin and mucous membranes in smallpox, bodies are found which vary in form, structure, and size. We regard these bodies as the parasites causing the disease. They occur within the epithelial cells, within the nuclei, and free. The forms within the nuclei are subsequent to those which develop within the cytoplasm. They are present in the greatest number in cases of the greatest severity and rapidity, of course. They do not occur as isolated structures, but one form follows another by gradual transitions, forming a cycle which corresponds with the cycle of living things.

"In the different cases the same forms are found at the same period of the disease. The bodies increase rapidly in the lesions, and the lesions increase in extent by continuous infection of adjoining epithelial cells. The same forms are found in corresponding situations in the lesions of different cases."

It is impossible to cultivate the parasite in artificial media and Koch's postulates cannot, therefore, be wholly fulfilled. Inoculation experiments in apes are very conclusive, for those animals are susceptible to both vaccinia and smallpox. The work of investigation is still being pushed, and further results may be expected in the near future.

*Predisposing Causes.*—It will surprise many to know that in former times smallpox was essentially a disease of childhood, over 80 per cent. of all cases occurring in children under five years. As vaccination is done chiefly in infancy and childhood, it is a strong proof of its efficacy that the occurrence of the disease has been transferred from infancy to adult life, when immunity has been exhausted. Susceptibility is almost universal, there being but few cases on record of complete insusceptibility. Unvaccinated infants and young children are particularly susceptible, but otherwise age and sex do not influence its transmission. Smallpox is most prevalent during cold weather. Extensive epidemics are not common during the summer.

*Source of Infection.*—Smallpox is directly contagious and may be transmitted by intermediary infection. The contagium resides in the exhalations from the lungs and skin, in various secretions and excretions, and in the pustules and dried crusts. The crusts dried and pulverized into dust may transmit the disease through clothing and breeding to great distances and render it almost impossible to completely disinfect a sick-room. The poison of smallpox is, in fact, the most tenacious and persistent of all the infectious diseases. There is considerable evidence to show that aerial transmission is possible through a radius of a quarter of a mile and perhaps more.

*Period of Incubation.*—The most common period is twelve days, the extremes being eight to sixteen days, though Osler asserts that there are authentic cases of twenty days' incubation period.

*Period of Contagiousness.* Smallpox is probably contagious from the first symptoms, and it certainly continues so until the last crust has fallen and all purulent secretions have ceased. The period of isolation can rarely be made less than six weeks.

*Clinical Types.*—Smallpox presents three types: the ordinary type (*variola vera*), which may be discrete or confluent; the hemorrhagic type; varioloid.

*Ordinary Type.*—This type runs its course in four stages: invasion, eruption, desiccation, and desquamation.

In children the onset is frequently marked by a convulsion and there may be several during the first twenty-four hours. In older patients a chill is the first symptom. Vomiting occurs early and may be many times repeated. It is accompanied by frontal and lumbar pain. The temperature rises rapidly and may reach 104° F. on the first day. The headache and backache are more intense than in any other infectious disease. On the fourth day and often earlier a macular eruption appears on the forehead and soon becomes papular. On the sixth day the papules change into vesicles filled with a clear fluid. On the eighth day the vesicles change to pustules and an areola forms. The pustules mature by the tenth day and soon rupture, begin to dry and form crusts. Desiccation goes on during the third week and desquamation begins. The crusts sometimes fall rapidly, but in other cases two weeks are required to complete this stage.

As the rash begins to appear the temperature begins to fall, but does not usually reach normal. The patient feels better and all the symptoms abate. As the pustular stage begins the temperature rises as high and often higher than did the primary fever. The symptoms return and there is great pain, particularly in the face, which is swollen so as to be unrecognizable. In the discrete type the secondary fever often begins to subside on the second day and reaches normal after ten days. In the confluent form there is often little or no remission after the primary fever, but remission may occur even in severe cases. The fever ranges above 104° F. and may persist through the third week. At the height of the pustular stage the patient presents a picture not equalled in any other disease. The face is a mass of pustules and if free areas are left they are inflamed and edematous. In fatal cases death occurs about the twelfth day. In other cases the disease subsides slowly, rarely by crisis. The symptoms gradually ameliorate and convalescence is established during the fourth week.

*Hemorrhagic Type.*—This type is not common in children. It occurs in three forms. In the first slight hemorrhages occur in the vesicles, which frequently abort and the disease usually runs a mild and short course. In the second hemorrhages occur in the pustules. The disease is severe and death occurs, as a rule, between the seventh and ninth days. Bleeding from the mucous surfaces may also occur. In the third form, the so-called *purpura variolosa*, a hyperemic eruption appears early, often on the second or third day, and frequently becomes hemorrhagic. Ecchymoses appear under the skin and conjunctivæ, the face is swollen,

PLATE XIX.



Smallpox in the Late Pustular and Desiccative Stage. Arms and hands show secondary umbilication due to rupture and central collapse of pustules. (Welch and Schamberg.)



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and hemorrhages occur from the mucous surfaces. Death occurs early, often before the appearance of the variolar eruption. It is to this form of the disease to which the name of black smallpox has been given.

*Varioloid.*—That form of modified smallpox which sometimes occurs in those who have been vaccinated some time before is known as varioloid. It is an unfortunate name, for it has sometimes led to carelessness in the isolation and care of patients. Varioloid is not simply a disease like smallpox, but it is smallpox. The clinical course varies considerably. Some patients are but slightly ill; others are seriously so. Generally the attack begins suddenly with symptoms common to the usual type, but less severe. The pain in the back and head may be severe and the temperature may go to 103° F. The eruption is scattered and abortive and does not pass through the full pustular stage. The temperature falls promptly and there is no secondary fever. The chief difference between varioloid and the ordinary type of smallpox is that in varioloid there is no pustular stage and no secondary or suppurative fever. The lesions simply come out as papules and in less than a week dry into warty or horny bodies which leave no mark.

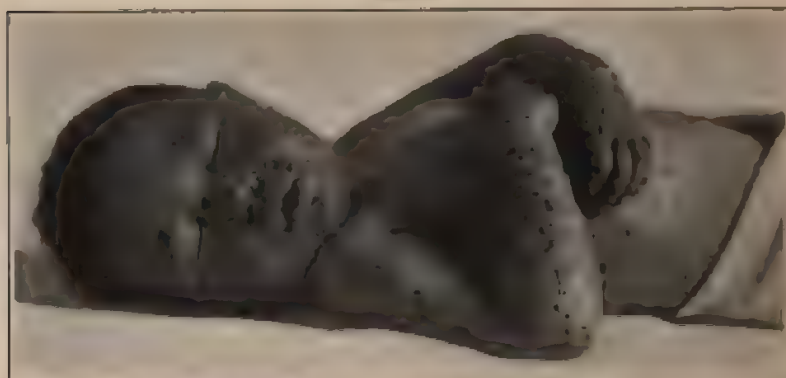
*Symptomatology. Fever.*—The onset of the initial fever is usually sudden and the temperature ranges high. It is often 104° F. or over on the first day and may go to 105° F. on the second and 106° F. or even more on the third. It falls as the eruption appears, usually on the fourth day. The secondary fever is at its height in favorable cases between the eighth and tenth days. It is at first somewhat remittent, but, as the case progresses, becomes more markedly remittent or actually intermittent. The temperature often falls gradually to normal during the third week of the disease, but this is sometimes delayed until the fourth week.

*Eruption.*—An initial rash sometimes appears during the stage of invasion. It may appear in a macular form simulating measles or as a diffuse erythema simulating scarlet fever. The characteristic eruption of smallpox appears on the third or fourth day. It is seen first on the forehead, face, wrists, and extremities, but in infants it often develops first in the folds of the skin, especially in the groin. It shows first as small, red spots, *macules*, of pinhead size, looking much like fleabites. On the fifth day these spots have become larger and darker in color; they are slightly elevated and may be felt with the finger as *papules*. The papules are tender and often the seat of burning pain. After twenty-four or thirty-six hours more the papules change into *vesicles*, each showing a clear summit with a slight depression, the so-called umbilication (Fig. 122 and Plate XIX.). By the eighth day the vesicles have become turbid and globular in shape, the umbilication having disappeared. These *pustules* are surrounded by a red areola and the adjacent skin is swollen and edematous and becomes painful. In weak and poorly nourished children the papules are sometimes pale in color and show very little areola. As the pustular stage is established the itching becomes intense and the patient, unless closely watched, tears his skin. When the pustules are fully formed there is a fetid,

sickly odor, and the patient, even in discrete smallpox, becomes a hideous object. The pustule being completed, it either ruptures and discharges its contents, which dries into a yellowish scab, or it dries down into a crust. This stage of desiccation begins on the face usually on the twelfth day of the disease. By the seventeenth or eighteenth day the stage of desquamation is established and the crusts begin to fall. They leave a depression stained a reddish-brown color, which gradually fades after five or six weeks. If there has been ulceration or a pustule has broken or the sore has been deep enough to involve the cutis, a white spot or pit is formed. The face slowly loses its purple color, but the pockmarks are permanent. In strictly discrete smallpox pitting is rare.

As the eruption appears on the skin it appears also on the hard palate and on the inside of the cheeks, and sometimes in the larynx and trachea. In the latter locations it is sometimes the cause of death in infants through associated edema. The eruption on the mucous

FIG. 122



Swelling of the face on the seventh day in a fatal case of smallpox. (Welch and Schramberg.)

surfaces pursues a different course from that on the skin. Each lesion begins as a papule of grayish color which soon ulcerates, leaving an excavated sore with a red areola. These ulcers are usually very sensitive and greatly increase the suffering of the patient. The eruption sometimes appears on the conjunctiva, leading to deep ulceration, followed by a permanent scar and sometimes by destruction of the eye. The lymph nodes of the neck are always more or less swollen when the throat is involved.

In confluent smallpox the lesions are close together; the inflammation and edema of the skin are excessive; as the pustular stage develops the face, hands, and feet become great ulcers. In no other disease does the patient become so transformed in appearance. He must be turned upon a sheet and handled with rubber gloves. Even in the most severe confluent cases the eruption remains discrete on the trunk. Sydenham pointed out long ago that the condition of the face is the best guide to the severity of the attack. He also laid down the rule that the more



the eruption appears before the fourth day the more liable it is to become confluent. The crusts in severe confluent smallpox are very slow in falling, a full month occasionally being required. In severe cases the confluent sores may be covered by large scabs under which suppuration goes on, destroying considerable areas of true skin. When these large crusts fall, broad scars are left which contract and may cause grave deformities.

**Constitutional Symptoms.**—In addition to the headache, backache, fever, and vomiting, which have already been referred to, there are sore throat, pain in the pharynx, restlessness, somnolence, and often delirium. The countenance has an anxious expression; the respirations are frequent and labored; the pulse is bounding and there is throbbing of the carotids; the face is flushed and the conjunctivæ are congested; there is great thirst. The spleen is enlarged and there is active leukocytosis. Muscular weakness develops with extreme rapidity. In severe cases a typhoid condition develops and the patient lies in a low, muttering delirium, with brown, dry tongue and all the symptoms of extreme nervous depression.

**Complications and Sequelæ.**—Unlike many diseases, it is the disease itself that is to be feared most in smallpox. Though febrile albuminuria may occur, nephritis is rare; this, however should not be overlooked. The only common complication referable to the digestive system is diarrhea, which is of most frequent occurrence in young children. Lobar pneumonia is rare, but bronchopneumonia is not uncommon. It is usually present in fatal cases. Salivation is rare in children. It commonly appears about the fourth day and lasts but three or four days. Suppuration in the joints occurs in rare cases. Orchitis and ovaritis are of not infrequent occurrence, but are usually of mild type. Laryngitis may be a serious complication. The accompanying edema may cause fatal occlusion or the cartilages may be involved by necrosis. Simple conjunctivitis is common in the early stages and is rarely serious, but the purulent conjunctivitis of the later stage is one of the gravest complications of the disease. The results are fortunately less serious now than they formerly were, when less care was bestowed upon the eyes. If the eyelids are kept from adhering and the eyes are cleansed frequently, excessive keratitis and perforating ulcers of the cornea can usually be averted. Otitis media resulting in perforation of the tympanum and otorrhea may prove a grave complication by causing deafness. Cellulitis and abscess of the subcutaneous connective tissue occasionally occur as well as local gangrene, and boils are very frequent during convalescence. Delirium is frequent during the febrile stage and post-febrile insanity sometimes occurs. Various forms of paralysis may appear, due probably to peripheral neuritis like that of diphtheria.

**Relapse and Recurrence.**—A peculiar secondary eruption sometimes occurs after desquamation, but it is a question whether it is a true relapse or not. Second attacks of smallpox are by no means unknown. Marson saw 47 second attacks among 5982 smallpox patients. Haeser states that in Verona 24 cases of second attack were observed within

ten years, and Hein reports 57 cases occurring in Württemberg between 1831 and 1835. This means that the period of immunity is not always lifelong.

**Diagnosis.**—The diagnosis of smallpox cannot be made, in the absence of known exposure, until the rash has begun to appear and can often be made with certainty only on the second or third day of the rash. The initial headache, backache, and febrile symptoms should put the practitioner on his guard, and he should isolate every suspicious case. The eruption on the first day is not characteristic and rarely becomes so before the second day. The presence of an initial rash has frequently led to error, a diagnosis of scarlet fever or of measles having been made. In typical cases doubt need not exist after the second day of the rash if due consideration is given to all the symptoms. As in all eruptive fevers, the diagnosis should not be made from the rash alone, but from the case as a whole. It is the cases of varioloid and mild smallpox, like those of the epidemic of 1900 and 1901, that lead to trouble. Such cases present some very difficult problems for diagnosis. The diagnosis between meningitis and smallpox is often difficult and sometimes impossible before the rash appears. The pain in head and back, vomiting, fever, and photophobia may occur in either. As a rule, the face in smallpox is flushed, while in meningitis it is apt to be pale and the fever does not range as high. Pustular syphilides may be mistaken for variolar pustules and pustular glanders may also be mistaken for smallpox. The disease most likely to be mistaken for smallpox is, without doubt, chickenpox. The differential diagnosis between these two diseases is considered under Chickenpox.

**Prognosis.**—The statement is frequently made that smallpox has become a much milder disease than it formerly was, and that vaccination, therefore, is less necessary. Facts certainly do not bear out this statement. It is quite true that during the recent epidemic the death rate has been low. It is true of all epidemic diseases that the prevailing type varies considerably in different years, and smallpox has certainly not shown itself of late in some localities in its most virulent forms. Among those who have never been vaccinated the disease in most epidemics is almost, if not quite, as dangerous as it ever has been. In the London Smallpox Hospital, between 1775 and 1800, all patients of course being unvaccinated, the mortality rate was 32.5 per cent. In 1853 Marsden found that the rate for the previous sixteen years was 35.55 per cent. of unvaccinated. In a recent study of smallpox Welch reports 1512 cases in unvaccinated persons, with a death rate of over 58 per cent. In young children the rate was much higher than this. Hart gives the death rate of unvaccinated patients as fully 40 per cent. In the Sheffield epidemic of 1887, the death rate was 54.2 per cent. It is certain that a death rate of over 50 per cent. is not abnormal for smallpox, including all ages and types.

The amount of eruption is the most important guide to prognosis; the greater the amount, the more grave the prognosis. Age is also an important factor. The mortality is very high in young children and

also in the aged. It is usually fatal under two years. Osler says that among 3164 deaths from smallpox in Montreal, 2717 were in children under ten years. Hemorrhagic smallpox is very fatal. The disease is especially fatal in the intemperate and debilitated. "Very high fever with delirium and subsultus are symptoms of ill omen." Death may occur in any stage. In malignant cases it sometimes occurs before the rash has developed. It sometimes occurs in children at the beginning of the suppurative stage, but is more common during the second week. It may then be due to complications, but is commonly due to exhaustion.

**Prophylaxis.**—One method of prevention is so pre-eminent above all others that it must receive chief attention. That method is *vaccination*. It is not only sufficient for preventing the occurrence of the disease in the individual, but if universally carried out in a community eradicates it. In view of the fact, however, that there is always in this country a considerable number of individuals who for one reason or another have not been vaccinated, the most stringent methods of isolation and disinfection should be enforced. The health officials are abundantly justified in forcibly removing smallpox patients to public hospitals where isolation can be made complete. Failure to promptly notify the health authorities of its appearance should be punished by heavy penalties. Where the disease does appear, the directions given for the management of scarlet fever should be enforced with the greatest thoroughness, and fumigation should be done with more than the usual care. Every city, in fact every community of any size, should have properly equipped hospitals for the treatment of smallpox, so arranged that they can be extended to accommodate the unusual numbers that they may be required to receive during an epidemic.

**Treatment.**—Treatment during the stage of incubation of smallpox is a question of very great interest, for it is possible to prevent the disease by vaccination if it is performed promptly. Welch, of Philadelphia, has had large experience in this direction and asserts that while no inflexible rule can be laid down, yet it may be said that if vaccination is performed on the first or second day after exposure, protection may be perfect, and if performed between the second and fifth days it may be partial. In vaccinia the areola appears between the seventh and eighth days and the sore is at its height on the ninth or tenth day. The incubation of smallpox is eleven or twelve days, and the eruption does not appear until the third or fourth day. Hence, the vaccinia has full opportunity to do its work. Welch bases his views on 194 cases of vaccination done during the incubation stage of smallpox. The accompanying chart (Fig. 123) demonstrates more clearly how the prevention of smallpox is possible by vaccination on the first day or two of the stage of incubation.

In the interval between the onset and beginning of the eruption, treatment is quite different from that required after the first week. Before the eruption begins to appear the treatment should be that of all severe febrile states. The fever is best controlled by the cold pack or the cold bath at 70° F. The severe pains can be relieved by nothing



but opiates, and they should be given without hesitation. Counter-irritation, and especially mustard pastes, should be absolutely prohibited, for the eruption is liable to become confluent upon irritated surfaces. The use of bismuth, lime-water, and similar remedies may sometimes mitigate the vomiting, especially in children, but they are usually less effective than the swallowing of small pieces of ice. The convulsions, delirium, and other nervous symptoms may be relieved somewhat by the use of bromide of sodium or potassium, or by chloral given by the rectum. Water may be given freely. The diet suggested for scarlet fever may be appropriately used during the febrile stage of smallpox.

FIG. 123

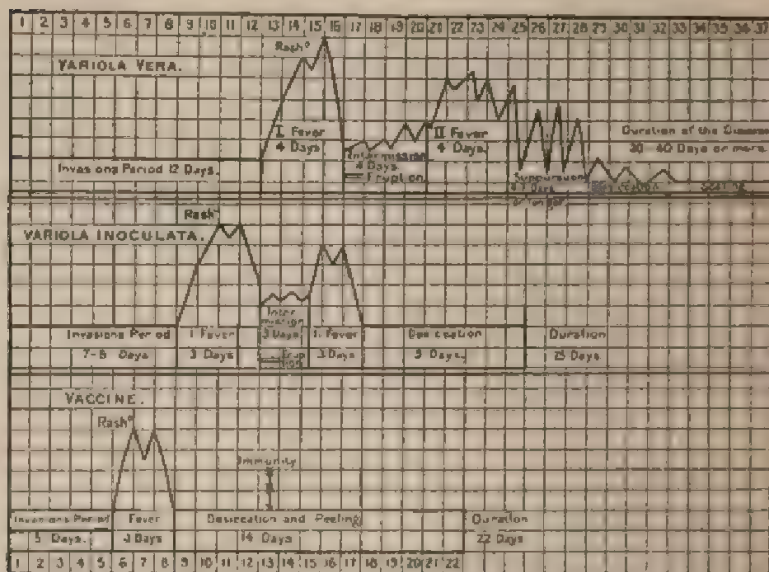


Chart showing the temperature range in typical cases of smallpox, inoculated smallpox and vaccine, and showing also the relative course pursued by these diseases. Vaccine being short in its invasion and course, active virus can be obtained from the pock on the fourth day and its immunising power is developed before the invasion period of smallpox is complete.—*British Medical Journal*.

In the later stages the appetite often improves and the stomach becomes more tolerant of food, so that the question of feeding is often not difficult even in quite severe cases. The tendency to overheat the patient is as strong in smallpox as in the other eruptive fevers and should be strenuously combated. The temperature of the room should not be over  $70^{\circ}$  F., and it should be well ventilated and the bedding should be light.

As the eruption begins to appear the fever subsides and the patient becomes more comfortable, so that many of the measures before taken may be dropped. Symptoms must be treated as they arise. The throat now usually requires attention. Bland washes, boric acid solution, etc.,



should be used and the mouth and throat should be kept as clean as possible without producing irritation. As the eruption develops, the burning of the skin demands attention; 2 per cent. carbolicized vaselin or a 2 per cent. ichthyol ointment may give relief. In some cases cool wet dressings may be more effective. As the eruption develops in the thick skin of the hands and feet it often gives great pain, which may be relieved by the application of ice-cold compresses. In some cases prolonged baths of the hands and feet in lukewarm water are more comforting to the patient. Very hot, wet, flannel bandages may even be used.

As the stage of suppuration begins the treatment must be again changed. Fever returns and the patient must face the dangers of suppuration. The physician must, therefore, give his attention to the fever. He must attempt to disinfect the purulent exudation, relieve the throat symptoms, and sustain the vital strength of the patient, for death from exhaustion is to be feared. The cold bath during this stage is difficult to administer and does not have the beneficial effect seen in many febrile conditions. During the pustular stage the itching is often intolerable. Scratching breaks the pustules, whose contents decompose and not only become offensive but poison the patient. Antiseptic treatment, therefore, is very important. Welch especially commends a mixture of olive oil and lime-water 15 c.c. ( $\frac{1}{2}$  oz.) and carbolic acid 0.65 to 1 c.c. (10-15  $\mu$ ). Oil of eucalyptus may be used in place of the carbolic acid. A great number of remedies have been proposed with the greatest assurance as capable of preventing pitting and disfiguring of the face. Both Welch and Osler, men of great experience, assert that not one has stood the test of extended use. No treatment can wholly prevent disfigurement in severe confluent cases. Any measure which will check or limit the depth of the inflammation will by so much limit the pitting. Some advantage is gained by protecting the face and hands from the light and air. Among the most efficient measures of treatment is the application of absorbent gauze soaked in cold water. Antiseptics may be added, of which carbolic acid is one of the best. The gauze may be cut into the form of a mask and should be removed as it becomes soiled. Crusts, when they begin to appear, should be kept softened with glycerin or cold cream. Later in the disease frequent baths are advantageous.

The eyes require treatment as soon as the lids begin to swell. When they show a tendency to become glued together, they should be separated every two or three hours and gently but thoroughly cleansed with boric acid solution or other mild wash. Constant care in keeping them open and cleansed will do much toward preventing blindness. Obstruction of the larynx should be watched, and, in extreme cases, tracheotomy should be performed. Diarrhea is best treated by some preparation of opium, especially paregoric. Little or nothing can be done for internal hemorrhage. Throughout this whole stage the strength of the patient should be supported. Stimulants and concentrated nourishment should be begun in all the graver cases as suppuration begins,

and should be pushed to their limit as soon as signs of exhaustion become apparent. The nourishment should be given at short intervals and in quantities which the stomach can tolerate. Welch gives not less than two quarts of milk each day in which two to four eggs are beaten, and he frequently administers six to twelve ounces of whiskey. Digitalis and strychnine in full doses may also be required.

During convalescence falling of the crusts may be hastened by a daily warm bath with carbolized soap. The patient should not be considered safe until every crust has fallen and all suppurating discharges have ceased. The tonic and stimulating treatment should be continued and a nutritious diet should be prescribed.

## CHAPTER XXII.

### CONGENITAL SYPHILIS—RHEUMATISM.

#### CONGENITAL SYPHILIS.

By GEORGE M. TUTTLE, M.D.

**ACQUIRED** Syphilis, either in infancy or before the age of puberty, is a rarity. Of course cases do occur from time to time, being inoculated accidentally, as a rule, but from a clinical standpoint they are in no respects different from syphilis as it occurs in adult life, and, hence, really need no separate description.

There is a form of syphilis, however, peculiar to infancy and childhood, differing in many respects from the acquired disease, and meriting careful consideration in every way. This is the inherited form of the disease, or, as it is ordinarily called, Hereditary or Congenital Syphilis. The terms are used rather promiscuously, or at least synonymously, to express the fact that the infection with the disease has taken place some time during either embryonic or fetal life, or it may even be in the time before the union of the ovum and spermatozoön.

There is little doubt that syphilis is an infectious disease of rather chronic nature, but as yet attempts to isolate a specific germ of the infection have been in vain. A number of micro-organisms have been isolated from syphilitic lesions by different observers, but no one of them has been accepted as the specific agent. One difficulty has been the impossibility of inoculating the disease on any animal, but within a very few months two different observers have announced the successful inoculation of the chimpanzee with syphilitic virus, and this observation may lead to the settlement of the disputed point by convicting one of these organisms or some as yet undescribed organism as the specific cause of syphilis.

**Etiology.**—Congenital syphilis may arise from infection through the spermatozoön, from the ovum itself, or from both coincidently, or subsequently to conception from the maternal tissues. A few cases of infection during the act of parturition have been reported, but these would scarcely come under the head of congenital syphilis. There can be no doubt but that the spermatozoa of a syphilitic father may convey syphilis to his future child. After the union of spermatozoön and ovum a father subsequently syphilitic can only infect an embryo indirectly through the medium of the mother.

On the other hand, a mother's possibilities for conveying syphilis begin with the germinal period, the unimpregnated ovum itself being syphilized, and continue through the embryonic and fetal periods of the intrauterine existence even to the time of birth. Of course, in case of syphilis in both parents the chances of the fetus being syphilitic are doubled.

If a woman contract syphilis while pregnant, she may, but probably will not, convey the disease to her offspring. The later in pregnancy the infection occurs, the less is the liability of fetal inoculation; but, clinically, the majority of cases escape the disease no matter what time it occurs, showing that the placenta acts as a protector to the fetus as well as the mother. This is not a universal law, but it holds good in most cases.

The father may inoculate a healthy ovum with a syphilitic spermatozoön, and a syphilitic child may be born of this conception, the mother, however, escaping the disease entirely; but she does acquire syphilis in a modified form as the result of having harbored a syphilitic fetus for nine months in her uterus. This is proven by the fact that she is able to suckle a child with syphilitic stomatitis without herself developing the disease, while another woman would be infected. This is the so-called "Colles' law." Some authorities insist that such immunity on the part of the mother can only have been acquired by her having had syphilis in a form so mild as to have escaped observation. Others, among whom may be named the great Joseph O'Dwyer, have questioned the existence of any immunity on the part of a woman bearing a syphilitic child. O'Dwyer used to state emphatically that a child born syphilitic could infect its mother if she were healthy, and would forbid the nursing of the syphilitic child either by its own mother or by any other woman on that ground.

Parents in the secondary or active stage of syphilis at the time of impregnation are almost certain to transmit the disease to their offspring; if in the tertiary stage, or where conception occurs after prolonged and proper antisyphilitic treatment, the danger of transmission is very slight.

**Pathology.**—The lesions of congenital syphilis may not be at all characteristic, but in the various viscera, and in the bones and skin it is common to find some rather typical changes, all of which partake, in a general way, of the nature of hyperplasias of the connective-tissue elements.

As would be naturally expected, since the placental blood enters the fetal circulation by way of the liver, the commonest visceral changes are found in this organ. The liver is usually, but not always, enlarged. There may be present in it rather widespread round-cell infiltration and general proliferation of the interlobular connective tissue. These fibroid hyperplasias follow in a general way the course of the smaller arteries. Accompanying this is a degeneration of the parenchymatous cells. In many such livers there are visible to the naked eye small, scattered, yellowish-white spots, the size of pinheads, giving the liver



a speckled appearance. These may be considered miliary gummata. The spleen is regularly enlarged, and the connective tissue being in excess, the spleen is harder and tougher than normal.

The lungs may show fibroid changes similar to those seen in the liver. There is an increase in the connective-tissue elements, without much or any change in the epithelium. Such lungs have a whitish color, and are tougher and less elastic in consistency than normal.

Even the kidneys may show evidences of some connective-tissue hyperplasia, with resulting parenchymatous degeneration, but such kidneys are in no wise characteristic of syphilis, and are found in other secondary conditions.

The lymph nodes may also present a moderate degree of small-cell proliferation, but are not characteristically invaded by the disease.

The osseous changes are more typical, and are more regularly present than many of the visceral lesions. There are ordinarily evidences of inflammatory changes at the junction of the shafts and epiphyses. These are commonest in the long bones, as the femur, tibia, humerus, or radius, but are quite frequent in the metacarpals, metatarsals, or phalanges, producing here the condition commonly called syphilitic dactylitis. In the milder cases, the medullary spaces are irregularly formed and the lime-salts consequently deposited in an atypical manner. In more marked cases the microscope shows real inflammation, with reddish or yellowish spots of osteomyelitis and proliferation of cartilage cells. This causes some enlargement and swelling of the epiphyseal junction, and in advanced cases we now and then find separation of epiphysis and diaphysis. These inflammatory processes may be confined to the neighborhood of one joint only, or may be seen coincidently in different bones.

In late cases we find osteophytic growths on the shafts of the long bones, due to a chronic proliferative periostitis, with the production of new bone under the periosteum. This leads to great thickening and enlargement and deformity of the affected bones, and is rather characteristically seen in the tibiae. Gummata may also form in the bones, and may break down and ulcerate just as in the tertiary form of acquired syphilis.

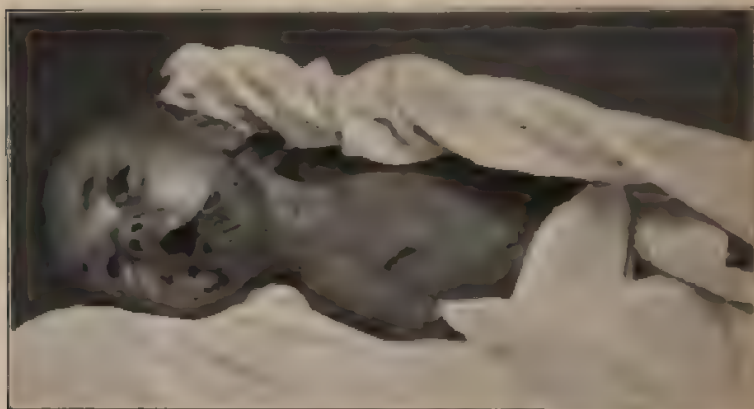
The skin rarely shows lesions commensurate with the clinical importance of the manifestations appearing in it. In most fatal cases the eruptions have disappeared and the skin shows nothing characteristic. If only an erythematous eruption has been present, nothing can be seen after death. On the other hand, we may see superficial erosions from bullous or pustular eruptions, or there may be distinct ulceration, especially about the anus or the mouth (Fig. 124). Microscopically, in the simpler forms of eruption we find simple round-cell infiltration, especially about the principal vessels and the glandular apparatus; in the ulcerative or pustular processes there will be more or less destruction of the epidermal layers of the skin.

**Symptomatology.**—The condition of the child at birth depends on two main factors, the virulence of the infection which it has inherited

and the stage of the disease in which it is born. For instance, the infection is so overwhelming in one fetus that it never comes to maturity, but miscarriage takes place at some period of intrauterine existence. On the other hand, many infants, the subject of congenital syphilis, are born to all appearances entirely normal, and only show mild evidences of the disease after a considerable time. Between these two extremes all degrees of development of symptoms are seen.

It must be remembered that in congenital syphilis there is no initial lesion to correspond with the chancre of the acquired disease, the infection having taken place through the fetal circulation, and that the period of incubation is of an indefinite length of time. Possibly the change of environment from a warm, fluid medium to a comparatively cool, gaseous one, which the skin and mucous membranes undergo at birth, is the exciting cause for the outbreak of symptoms. At any

FIG. 124



Infantile syphilis; eruption and fissures of mouth. (Gottlieb.)

rate the average infant the victim of congenital syphilis is born normal, and the most careful inspection will fail to find any sign of the presence of the disease. The real evidences of the disease begin in a very large percentage of the cases during the second, third, and fourth weeks of postnatal life. Occasionally they are postponed until the second month.

Accordingly the clinical history of congenital syphilis may be: miscarriage during the early months of pregnancy; the birth, prematurely or at term, of a dead fetus showing undoubted lesions of inherited syphilis in its skin, bones, and viscera; rarely the birth of a living infant in the eruptive stage of the disease, showing lesions of the skin and mucous membranes; but usually, if the pregnancy go to term, a living child showing no evidences of syphilis.

Our consideration of the subject does not include the study of the first two varieties—stillborn infants.

In cases born with the disease fully developed we find a decided

degree of malnutrition present. The infant is emaciated, its skin wrinkled, and its appearance that of senility. Skin eruptions are the most characteristic evidences of the disease. Vesicular eruptions seem the most common, and these are regularly seen on the palms and soles as a palmar or plantar pemphigus. The vesicles may contain purulent serum, and may have burst, leaving a loose torn skin hanging, attached at the edge. In other places they may dry up and form yellow crusts on various parts of the body. A coryza may be present at birth, and the mucous membrane of the lips may be shiny and dry, and tend to crack. These infants are regularly very feeble, and usually live but a short time.

In the ordinary case of the birth of a normal-appearing infant, the clinical history is quite different. On close inspection such an infant will show a clean skin and normal mucous membranes, but there is often a little anemia present, which if watched gradually increases. Some of these infants are liable to suffer from hemorrhages at the umbilicus, and the cord is often friable. The infant may become wakeful and its nutrition begin to fail; but the first characteristic sign that shows itself is a persistent coryza, due to mucous patches, that appears about the third week—the so-called “snuffles.” The nasal discharge may be profuse or only moderate; it may be thin and watery, or at times a little blood stained, but it does not respond to the ordinary means that are used to cure such discharges. Accompanying this condition the infant’s cry may be hoarse, and inspection may show mucous patches in the mouth and throat. The lips may be fissured more or less, producing rhagades (Fig. 124), which leave the tell-tale, radiating scars of later life (Fig. 125).

Almost coincident with these lesions in the nasal and oral mucous membranes appear the typical cutaneous manifestations of the disease. As in the acquired disease, the skin lesions are multifiform and may present a variety of lesions at the same time. Thus we may see a simple erythema or roseola at one time, or macules, papules, vesicles, or even pustules at another.

The most common eruption is the maculopapular syphilide. The infiltration in the skin may be almost *nil* when only macules are present, while at other times or in other places it may be rather extensive, causing decided thickening and a resultant papular condition. The macules and papules show a decided tendency to coalesce, producing a continuous eruption in places, while the outlying areas present normal skin between the individual lesions. These confluent rashes seem to be distributed especially where the irritation is greatest, and are frequent about the buttocks and genitals, and generally extend down the thighs on to the calves and on to the feet (Fig. 126). The face is another common situation for these macular eruptions.

The margins of the continuous eruptions are irregular, and often separated by clear skin from scattered but smaller patches of the same kind of rash. The edges are usually but slightly raised above the level of the sound skin, but may be decidedly infiltrated and thickened.

The epithelium may in places peel off and leave ragged edges, this is seen oftenest about the feet and hands. In the early stages these macules and papules are bright red, but as they grow older they grow duller in color and finally become copper or ham colored, which is considered rather typical of syphilitic rashes.

Vesicular syphilides are rare, but may occur. On the palms and soles they produce the well-known pemphigus. The vesicles vary in size from a pea to the larger bullæ, which may cover a good-sized

FIG. 125



Results of congenital syphilis, showing scars around the mouth. (Stowell.)

of skin. They contain more or less serum, which may be reddish in color. On rupturing, the underlying skin is seen to be infiltrated and copper colored.

Pustular eruptions are common and are often mixed in with maculopapular rashes. They occur mostly on the face, head, and buttocks, but may appear anywhere. They often come in groups about the forehead or near the anus. On discharging their contents and drying, crusts are formed. Very probably the pustules are most frequent



due to infection of pre-existing syphilitic rashes by pus-producing organisms.

Alopecia is ordinarily present to a greater or less degree, but is scarcely characteristic, as it occurs in so many forms of malnutrition. This may involve the eyelashes and eyebrows, as well as the scalp. The nails

FIG. 126



Case of congenital syphilis. (Gottlieb.)

may show signs of inflammation at their junction with the skin, and if the matrix is involved the nail may be shed.

The mucocutaneous junctions are usually the seat of lesions. Those of the lips have been already described. The anal orifice often shows mucous patches, ulcerations, fissures, or condylomata, depending on the severity of the individual lesion. The same is true of the vulvar orifice (Fig. 127).

As in most bone diseases occurring during growth, those of syphilis

are also commonest about the epiphyseal junctions. The symptoms are pain, tenderness, and swelling, but very little redness. Resulting from these changes, the joint is more or less voluntarily immobilized, as movement increases the pain, and a pseudoparalysis may be produced, for which often these babies are brought to the physician. Pseudopar-

alysis may occur without separation of the epiphysis. The bones of the upper or lower extremities are most likely to be involved, such as the humerus or femur, but any bones may be attacked, even the clavicles or the ribs.

Syphilitic epiphysitis may occur singly or be multiple, and is often quite difficult to distinguish from that due to rickets or tuberculosis. Often only treatment will settle the diagnosis. The epiphysis may separate from the shaft, but the skin is seldom involved and sinuses are rare. The neighboring joints usually escape involvement, although a secondary arthritis, probably due to some pus-producing organism, may occur as a complication. Syphilitic dactylitis involving the phalanges and metacarpals or metatarsals may occur, and is very similar to the condition produced by tuberculosis. Here we often find necrosis and sinuses resulting from the extrusion of dead pieces of bone. Peri-



FIG. 127  
Infantile syphilis, large area of condylomata on buttocks. (Gottlieb.)

ostitis of the proliferative variety may occur, causing thickening in the shafts of the long bones, and often the formation of nodes on the flat bones, such as the frontal or parietal bones—the so-called cranial bosses. These may rarely suppurate.

On examining the abdomen of infants presenting any or all of the above signs, we regularly will find the liver and spleen palpable well below their usual location and distinctly enlarged. The edges of both will be clean cut and give the impression of hardness. Except in the presence of some skin or bone lesion draining into the neighboring lymph nodes, the nodes will not be found so uniformly and generally enlarged as they are in the acquired disease.

Gummata may develop anywhere in the body. In the skin, if untreated, they break down and form ulcers. In the mucous membranes, as those of the nose and mouth, they regularly invade the bone, and as they ulcerate they may perforate the nasal septum or the hard palate.

or may cause necrosis of the nasal bones. These perforations of the nasal septum or roof of the mouth are quite typical. When involving the nasal bones they lead to the deformity known as saddle-back nose, a distinct depression at the junction of the nasal and cartilaginous portions. Gummata may likewise occur in the viscera, but are ordinarily not diagnosed in these locations. In some late cases paresis of one extremity with symptoms simulating lead palsy—*e. g.*, drop-wrist—may develop.

Anemia and all the evidences of malnutrition gradually increase during the course of the development of the symptoms, and from time to time an irregular fever may be present.

The child may die of wasting, or of some intercurrent disease, but if the infection is mild, and if proper and energetic treatment is pursued, the evidences of the disease, the secondaries, as they really are, gradually disappear and the child may completely recover.

One of the results of an early keratitis is a corneal opacity. The permanent teeth often present rather a characteristic appearance, the so-called "Hutchinson's teeth" (Fig. 128). In this condition the upper central incisors are deeply notched by a crescentic depression in their cutting edge, the enamel is imperfect, and the teeth themselves are shaped like a peg and rounded. The ear having been involved, a chronic otitis of the inner ear develops, causing a loss of conducting power in the auditory nerve. The stigmata which follow early syphilis, interstitial keratitis, pegged and notched teeth, and deafness, are sometimes called Hutchinson's triad, and are considered quite pathognomonic. The most important of these signs are the so-called Hutchinsonian teeth.

**Diagnosis.**—In case of stillborn infants the presence of bullæ and the examination of the viscera usually will clear the diagnosis. Maceration must not be considered a syphilitic symptom.

In living infants a well-marked case presents few difficulties. The parental history must be taken into consideration, particularly the results of previous pregnancies of the mother. Repeated abortions and the subsequent birth of a living child presenting some signs of the disease are suspicious circumstantial evidence.

An incurable coryza, fissures about the mucocutaneous regions, epiphysitis, multiform rashes, condylomata, and malnutrition are all valuable signs. Enlarged spleen and liver are confirmatory. Rachitis presents a number of these same signs, but it must be remembered that syphilis produces its effects usually during the first half of the first year of life, while rickets shows most plainly during the latter half of the first year and the first half of the second. In other words, rickets takes time to develop, while congenital syphilis begins to present symptoms soon after birth.

Hutchinson's triad, or any two of the three signs, are very typical. The bony lesions are very difficult to separate from similar changes due to tuberculosis. Often only a therapeutic test will decide the question. Deformities of the nose and hard palate are always aids,

and likewise thickenings and deformities of the tibiae. Paralysis due to syphilis is not symmetrical and will yield to specific treatment.

**Prognosis.**—This is a much more fatal disease than acquired syphilis of adult life. Malnutrition is a frequent cause of death, and the lowered vitality due to this cause makes such infants very susceptible to other diseases; they often die of complications that a normal infant would survive. The earlier after birth the symptoms develop the worse the

FIG. 128



Hutchinson's teeth. (Stowell.)

prognosis, and naturally the earlier and more vigorous the treatment the better the chances for survival.

Even if they escape death through a mild infection, or as the result of active treatment, children the victims of this disease never seem to develop entirely normally. They show evidences of malnutrition of one kind or another, and they often develop rickets. Their growth may be stunted, their mentality may never be all that it should, and finally we often, even after most careful treatment, see some of the late



signs of the disease developing about puberty. So that in no way can the prognosis be looked on as other than unfortunate.

**Treatment.**—If a woman known to be syphilitic should become pregnant, or if a woman should be pregnant by a man known to be syphilitic, antisiphilic treatment should be begun as early in pregnancy as practicable, and should be continued until the beginning of labor. This treatment should be the ordinarily accepted one by mercury or iodide of potassium, or both combined, according to the indications for the individual case. If a child is born to such a mother, every effort should be made to aid natural breast feeding, as breast-fed children always do better than hand-fed ones when subject to this disease. During lactation the antisiphilic treatment of the mother should be continued whether she seems to need it or not. No wet-nurse should ever be employed for a syphilitic infant.

Immediately on making a diagnosis of congenital syphilis mercurial treatment must be begun. It may be administered through the skin by inunction, or by way of the stomach. It is often wise to use first one method for a time and then to change for a period to the other. Children bear mercury well and seldom suffer from salivation, but their bowels easily become loose, and this is always an indication for the use of the inunction method.

For inunction the ordinary blue ointment, *unguentum hydrargyri*, is used in daily dose of about 1.3 gm. (a scruple). The location for rubbing the mercury in should be changed from day to day to avoid irritating the skin. The soles of the feet, the skin of the thighs or upper arms, and the surface of the back or abdomen can be used one after the other, or it may be put under the binder. This method is sure, but dirty and troublesome. The oleate of mercury from 0.5 to 5 per cent. strength is sometimes useful and it is a cleaner preparation than the blue ointment. Resorbin is a good vehicle for mercury.

The best preparation for internal use seems to be "gray powder," *hydrargyrum cum creta*. The chalk serves to counteract slightly the laxative tendency of the mercury. It should be given in doses of 0.065 gm. (1 gr.) three times a day. As the child grows older, or in the cases of severe infection, 0.130 gm. (2 gr.) three times a day may be given. Some physicians give calomel in 0.0065 gm. ( $\frac{1}{16}$  gr.) doses three times daily; others the corrosive chloride in 0.00108 gm. ( $\frac{1}{80}$  gr.) doses three times daily; and others the protiodide, *hydrargyrum iodidum viride*, in 0.0162 gm. ( $\frac{1}{4}$  gr.) doses three times a day. With any of these preparations for internal use, in case of diarrhea, a little opium may be combined, as Dover's powder in 0.0162 gm. ( $\frac{1}{4}$  gr.).

Locally, fissures, ulcers, and condylomata should be treated by dusting with dry calomel powder, or by the application of calomel ointment, in the proportion of 4 gm. (1 dr.) to 30 gm. (1 oz.) of vaselin. In severe skin lesions a daily bath in 1:20,000 solution of corrosive sublimate may be valuable. "Snuffles" is treated by cleansing the nose with a mild alkaline wash, as Seiler's solution, and then smearing the inside of the nostrils with *unguentum hydrargyri ammoniati*, or the

calomel ointment. Later treatment will require iodide of potash in the saturated solution, dose, 0.06 c.c. to 3 c.c. (1 to 5 drops) three times daily.

Treatment should be continued without interruption until all signs of the disease have disappeared, including enlarged liver and spleen. After that interruptions can be made in it from time to time, with renewal of the treatment again for the two to three years that are ordinarily looked on as the proper length for continuing in the acquired disease.

Everything possible in a hygienic way should be done for these infants: regular hours for eating and sleeping, abundance of fresh air, scrupulous cleanliness, and, in case the mother cannot nurse her infant, careful attention to all the special rules for scientific artificial feeding. An iron tonic may be of great value as an aid.

### RHEUMATISM.

By JOHN RUHRÄH, M.D.

Rheumatism is an acute, non-contagious fever, the exact cause of which is as yet unknown. In children over ten years of age it is characterized frequently by the same symptoms as are seen in adults: fever, multiple arthritis, great pain, a tendency to involvement of the fibrous tissues, and of inflammations of the heart, and sour sweating. In children between five and ten, and even younger, typical attacks of articular rheumatism may occur, but atypical attacks are much more common. Under five years articular rheumatism of the adult type is very rare and the disease is manifested by a number of symptoms no one of which can be regarded as pathognomonic, but which, taken together, form a symptom-complex that makes the diagnosis possible.

The variations of the disease as seen in infants and young children make it liable to be mistaken for other diseases, while, on the other hand, totally different affections are called rheumatic.

**Etiology.**—The exact cause of the disease is unknown, but there are numerous theories. Three of these may be mentioned:

1. That rheumatism is an infectious disease. This is borne out by a study of the occurrence of the disease. It may be seen in epidemics and these epidemics are liable to be followed by outbreaks of less severity. It occurs, of course, apart from any epidemic. Poynton and Paine, Triboulet and Wassermann have independently isolated a diplococcus, nearly identical in each instance, which they regard as the cause of the disease, but this is not yet confirmed. The infection theory, while not definitely proven, is generally accepted.

2. That the disease is due to chemical or metabolic causes. This is based on the idea that there is defective assimilation, with the formation of abnormal products, which are toxic. Lactic acid is the most frequently mentioned of these.

3. That the disease has a nervous origin.

**Pathology.**—There are no characteristic postmortem changes. The joints when affected are swollen, the swelling affecting the synovial

membranes and ligaments and the surrounding tissues. There is hyperemia and a not very abundant effusion which is somewhat turbid and contains leukocytes and some flakes of fibrin. The pleurisy and pneumonia which are frequently found are due to other organisms and there is nothing distinctive in the lesions. Changes in the heart are mentioned on p. 695.

**Occurrence.**—The disease is most frequently found in the temperate climates where the humidity is high. It affects girls somewhat more frequently than boys. This sex difference lasts until about twenty years of age. It has been thought by many to be an hereditary disease. It certainly seems to occur in families, but whether that is due to transmission of a tendency to have the disease or whether, as may be probable, it is due to house infection or to house occurrence cannot be definitely stated. It is quite certain that exposure to cold and wet predisposes one to an attack. One attack does not produce an immunity, but rather predisposes to a second.

**Symptomatology.**—The adult type is usually seen in children from about ten years of age. The older the child the more liable the disease is to conform to the adult type, and the younger he is the farther the symptoms will deviate from it. In the younger cases there are several marked differences. Not many joints are involved. The pain is not so severe, nor the fever so high; the sweating is not marked and has little or none of the characteristic sour smell as observed in adults. Instead of lasting three weeks or more the attack is usually over in two weeks or even a few days. Relapses are uncommon just as recurrences are frequent.

In acute cases in children the onset is usually sudden, with no prodromes. There is more or less pain in one or more of the joints; there is fever, which is ordinarily not very high and may only be 100° or 101.5° F., although it may reach 105° F. There may, however, be a gradual onset, with vague pains in several joints; an indefinite condition of malaise, with or without some of the other manifestations of the disease. Usually these cases, sooner or later, show more or less marked symptoms of a definite attack. A rarer mode of onset and one which may be very puzzling is to have fever, headache, and some gastric disturbance for several days before there is any pain. There may be a tonsillitis at the outset, or, after a day or two of indefinite symptoms, heart murmurs may be made out.

The joints most usually affected are the knee, ankle, small joints of the foot and the wrist, but no joint can be said to be exempt. The joints may be swollen without much pain, and if in the upper extremity may be overlooked. The pain may not be severe enough to keep the patient in bed or it may be as severe as in the adult type of the disease.

The symptoms persist for varying lengths of time. In some cases of rheumatism the patient is all right in from five to seven days; the average case lasts about two weeks, while some may drag along and tend to become subacute or chronic.

In children under seven, attacks of articular rheumatism are rare, but other manifestations may be found. Under three rheumatism is



rare, but it has been observed even under one year of age. In these young children the swelling of the joints may be slight and transient, with little or no heat or redness. In younger children, and in the older ones as well, the symptom-complex is made up of a number of things, and these are seen in divers combinations, and sometimes one symptom is prominent and sometimes another. The course is extremely variable. In acute cases, however, it is liable to be about two weeks.

*Heart Lesions.*—These are described in detail in the section on Diseases of the Heart, but the subject warrants the following brief mention. Involvement of the heart is a frequent occurrence in rheumatism in children. It is said to be more liable to occur in them than in adults. There is either an endocarditis, a pericarditis, or a myocarditis. (See p. 695.) In all cases of suspected rheumatism the heart should be carefully studied each time the patient is seen. It is a notorious fact that acute pericarditis and endocarditis are very frequently overlooked. When found in the course of a febrile disease of obscure nature, with some of the symptoms as outlined below, it is quite safe to assume that it is rheumatic and the child should be treated accordingly. In most cases of chronic cardiac disease in children there is a history of rheumatism or of vague joint or muscle pains which may not have been correctly diagnosed when observed.

*Chorea.*—This is another frequent manifestation of the disease. The relation of chorea and rheumatism is discussed in the chapter on Chorea. It usually comes on after an attack of rheumatism. In about half the cases of chorea there is a history of rheumatism. Sometimes it may precede the other symptoms of the disease. In still others the child is choreic and when articular symptoms appear the chorea gets better to recur on the subsidence of the joint trouble.

*Tonsillitis* is frequently associated with rheumatism. This applies to all forms of tonsillitis and to pharyngitis. An acute attack of rheumatism may begin with tonsillitis. Frequent attacks of tonsillitis in a child should always be regarded with suspicion; and if there is associated heart lesions or other manifestations of rheumatism, there can be but little doubt as to the nature of the throat trouble.

*Skin lesions* occurring in the course of rheumatism may be regarded as a part of the disease. These are so varied that they are most easily described as erythema multiforme. Sudamina are common. There may be erythematous rashes not unlike a scarlatina rash. Red miliary rashes are frequently seen. Erythema nodosum is also met with. In this latter affection there are a number of nodules from the size of a bean to a pigeon's egg, mostly over the anterior part of the leg and particularly over the tibia, but occurring on the face and other parts of the body as well. These are at first red and then change to a purple or bluish color like a bruise. They are tender and painful. Their duration is about two or three weeks. Purpura is not quite so frequent in children as in adults, but is met with. There are severe forms of purpura described under the name of peliosis rheumatica where there is fever, swelling of the joints, bleeding from the gums and mucous



membranes and enlarged spleen. There is a reasonable doubt as to whether these are really rheumatic or not.

*Subcutaneous nodules* were described by Barlow and Warner. They are more common in England than in this country, but are met with here and their rarity may be in part explained by the fact that we are not so in the habit of looking for them as they are abroad. They are of considerable diagnostic value when they are present. (In England they are found in about 20 per cent. of the cases.) They consist of small transitory nodules which have been described as fibrous, and they feel like it, but are really only a transient infiltration of the tissue. They vary in size from a pinhead to a split pea, and are found over the bones which are covered only with skin and subcutaneous tissue, about the joints, and along the course of the tendons. They are found especially on the hands and wrists, on the olecranon, about the patella and the malleoli, along the spine of the scapula, and on the vertebræ. While somewhat difficult to see, the nodules may be easily felt. They come and go, remaining weeks or months. They are neither painful nor, as a rule, tender. They are more frequent in children than in adults. They may come on early in the disease or about the time the patient is getting well, or they may come on without any acute symptoms. They are even associated with heart lesions. Similar or nearly similar nodules are found in arthritis deformans, gout, and migraine.

*Pleurisy and pneumonia* are both frequently met with in the course of rheumatism, but may be regarded as true complications, being due to mixed infections.

*Nervous symptoms* are common in rheumatic children. Even if there is no chorea, there is liable to be a nervous condition—a sort of hypersensibility. The children start at noises, make nervous, purposeless movements, grimaces, and the like. They are also prone to headaches and to nightmare. Delirium or even coma may be met with in the severe forms of the disease. Meningitis of rheumatic origin has been described, as has also neuritis.

*Anemia*.—Almost all children who have had rheumatism have a more or less severe grade of secondary anemia. This follows so regularly that anemia has been included as one of the features of the disease.

*Muscular rheumatism* undoubtedly occurs in children, although some have stated that it is rare. The growing pains so frequently complained of may in part be explained in this way. A child with "growing pains" should be examined carefully for other manifestations of rheumatism. A form most frequently seen attacks the muscles of the neck producing torticollis. In these cases there may be more or less fever, following a rather sudden onset. The muscle is tender to the touch and usually very painful. The muscle is in a state of contraction and the patient either cannot or will not move it. The symptoms are less severe when the patient is in bed. The head may be twisted to one side or be retracted according to the muscles affected. These cases may roughly simulate a meningitis, but the diagnosis is usually easy. They may also be mistaken for caries of the spine and the diagnosis be rather

difficult; the duration of the disease clears up the case where there is any doubt. The muscles of the legs are next frequently affected and almost any group of muscles may suffer. The duration is usually about a week, but some of the cases may last much longer. There is frequently associated tonsillitis and heart lesions.

**Diagnosis.**—The diagnosis of rheumatism in infants and children is not always an easy matter. True rheumatism is frequently overlooked and what is called rheumatism is often some other affection, and *vice versa*. It is important to make a correct diagnosis as the future welfare, the life itself even at times, may depend upon it. This is true where infected, suppurating joints are mistaken for it and also in the case of scurvy.

The symptom-complex as described above must be borne in mind. In doubtful cases much can be inferred from the family history and the previous history of the child. The previous occurrence of skin lesions, of tonsillitis, or of joint or muscle pains all have some weight in deciding the question. The presence of these or of a heart lesion is of the greatest importance.

Acute rheumatism must be differentiated from the following diseases:

*Scurvy*, many cases of which are called rheumatism. The infrequency of rheumatism during the first two years of life, and the comparative frequency of scurvy in artificially fed children should arouse a suspicion of the latter disease where there are obscure joint pains in an infant. The nature of the food of the child, the presence of swelling or bleeding of the gums, the subperiosteal hemorrhages giving rise to large swellings about the long bones, and the absence of fever are all valuable points. The hemorrhages into the skin in scurvy are apt to be spots from half an inch to two inches in diameter and look more like bruises than do the purpuric spots of rheumatism. They are also usually near or about the larger joints. It must be borne in mind that scurvy may occur without gum lesions or hemorrhages; in fact, any part of the symptom-complex may be wanting. When in doubt a proper diet and orange-juice will clear up the diagnosis in most cases in a few days.

*Rickets* may be easily mistaken for rheumatism. The restlessness, sweating about the head, throwing off the covers at night, rickety rosary, craniotabes and the nature of the feeding will generally prove sufficient to differentiate the two diseases.

*Multiple secondary arthritis* occurs after a number of diseases among which gonorrhea, dysentery, scarlet fever, and cerebrospinal fever are the most important. In infants and young children the gonorrheal form may be met with as well as in later life. Clement Lucas found 23 cases, 18 of which followed ophthalmia neonatorum. One or more joints may be affected after any of the diseases mentioned. The chronicity of the disease and the fact that it occurs in the course of one of the above-mentioned diseases serve to differentiate it from rheumatism. In gonorrhea the diplococcus has been found in the joint by aspiration. This is also true of the meningococcus in the joint troubles of meningitis.

*Septic arthritis* is really a form of the above, being a septic infection of the joint. In many cases there is a definite source of infection, an abscess, or something of the kind. In some cases the original source of infection cannot be found or is overlooked, and the joint is the first thing noticed. The diagnosis can usually be made from the fact that the temperature is liable to be higher and both local and constitutional disturbance very much greater than found in rheumatism.

*Acute osteomyelitis* is also important, for if the diagnosis is not made and the bone opened, death is liable to result. There is high fever, serious constitutional disturbance, and the swelling is above rather than about the joint. *Acute arthritis of infants* is usually seen in rather young infants. There is a rapid effusion into the joint, which either starts as a purulent effusion or rapidly becomes so. The knee or hip is usually the joint affected, but the elbow and other joints may be. As a rule, but one joint is affected. The size of the effusion, the high fever, marked local and constitutional disturbance make the diagnosis possible. When in doubt it is better to use an aspirating-needle than to wait, as the joint may be rapidly destroyed if not drained. *Acute articular gout* is not seen under seven years of age. The presence of gouty deposits and a careful examination of the urine and marked heredity might make the case clear in older children. Many indefinite symptoms not unlike rheumatism have been described in the children in gouty families. The effect of diet on these is of some help in determining their nature. Effusion of blood into the joints sometimes occurs in bleeders. (See article on Hemophilia.) The fact that the child is a bleeder and the nature of the effusion, the absence of fever (not always absent, however), and of other symptoms help in the diagnosis.

In this volume some of the diseases liable to be mistaken for *chronic rheumatism*, as well as that condition, will be gone over with the differential diagnosis under each. It should be remembered that rheumatism itself rarely leaves any permanent joint changes. This is especially true in early life. Where there are chronic joint changes with grating, abnormal deposits and the like, it is almost certain that the disease is not rheumatism. The importance of bearing this in mind is to avoid in such cases the constant use of antirheumatic remedies which do not influence the other diseases and which in themselves may be prejudicial to health if kept up for any length of time.

**Prognosis.**—In children this is in a general way good. Barring complications the child is certain to recover, but one can never be sure that there will not be involvement of the heart. Each attack of rheumatism predisposes to another attack.

**Treatment.**—The treatment of acute rheumatism is not as satisfactory as it might be; pain can usually be relieved and the patient rendered comfortable, just how much the disease can be cut short, if at all, is a question, and we have no way of preventing the heart from being affected.

The child should be put to bed and kept there until all traces of the disease have disappeared. It is better to keep the patient in bed a few days too long than to let him up too soon. He should be carefully



guarded against chilling and should be between blankets or have long flannel gowns. When he gets up he should wear flannel underwear and be kept out of the damp, cold, and draughts. The *diet* which is best suited to rheumatism is a matter of some difference of opinion. Some authors recommend farinaceous foods, and others, notably Holt, recommend the use of nitrogenous food and the restriction of the starches. While the child has fever the diet should be largely composed of milk. This may have lime-water or Vichy added to it, or barley or oatmeal gruel. Broths, custards, junket, and gruels may be used to vary the diet. If the urine is examined daily it will show the influence of diet. While there can be no doubt that the average febrile rheumatic patient does better on a non-nitrogenous than on a nitrogenous diet, there are patients who have subacute symptoms who seem to require meat in their dietary. Urine that is persistently acid may be caused in such cases by an intestinal fermentation and the use of too large a quantity of farinaceous food, so that meat simply serves to lessen the intestinal indigestion without apparently increasing the nitrogenous waste. When the fever disappears milk should still be used as much as possible, but the green vegetables may be added, and meats and eggs may be given, but they should not be allowed with acute symptoms or where there is any question of nitrogenous excess. The food should then be easy of digestion and of good quality.

*Therapeutics.*—Locally I have used Fuller's lotion as recommended by Osler. This consists of carbonate of soda, 24 gm. (6 dr.); laudanum, 30 c.c. (1 oz.); glycerin, 60 c.c. (2 oz.); and water, 270 c.c. (9 oz.). It should be applied hot on flannel cloths. Chloroform liniment may also be used. If the joint is very painful, it should be fixed by wrapping it in a generous roll of cotton and bandaging to a well-padded splint. In older children the joint may be "fired" by using a Paquelin cautery, which should be heated to a dull red and passed to and fro rather rapidly, just close enough to the skin to cause a glossy redness to appear. Great care should be taken not to produce any definite burns. With a little practice this is easily done and affords great relief.

Internally salicylic compounds may be used or alkalies. Of the former there are a number to choose from. Salicin is one of the best for children. It is usually better borne than the others and is not so depressing. It may be given in doses of about 0.06 gm. (1 grain) for each year of the child's age, and this amount should be repeated every hour or every two hours until the pain is relieved. After that it may be given every three or four hours and later on the dose reduced in size.

Salicin is inodorous, but very bitter, with the peculiar flavor of the bark. In older children it may be given in capsules. It is not well to make the capsules larger than 0.26 gm. (4 gr.) each. If the child cannot swallow a capsule it may be given in some flavored syrup. Salicin is soluble in about 30 parts of water.

℞—Salicini	1.56 gm.	(gr. xxiij).
Syrupi ananasi	60.00 c.c.	(3ij).

Sig.—A teaspoonful every two hours. From one to two years of age.



Salicylic acid is almost insoluble in water, is tasteless, but leaves a sense of bitterness and astringency in the mouth. It is quite efficacious in stopping pain, but it may be very depressing. The pulse rate may be greatly decreased by its use. The dose is about 0.03 gm. ( $\frac{1}{2}$  gr.) for each year of the child's age. It may be given in tablets or capsules. The following has been advised as a pleasant way of giving it in solution:

℞—Acid. salicylici . . . . .	4.0 gm.	(3j).
Oil. amygdal. express . . . . .	20.0 c.c.	(5v).
Pulv. nucis . . . . .	10.0 gm.	(5℥ss).
Syr. amygdalæ . . . . .	24.0 c.c.	(5vj).
Aq. aurantii florum . . . . .	q. s. ad 90.0 c.c.	(3℥j).

Sig.—A teaspoonful every two or three hours.

The salicylate of soda is quite soluble (in 1.5 parts of water), but may be rather nauseating. To young infants it is best given in plain aqueous solution or with a flavored water, as peppermint- or cinnamon-water. For older children it may be put up with syrup of orange, syrup of raspberry, or aromatic elixir.

Oil of wintergreen, consisting almost entirely of methyl salicylate, may be given in place of any of the above. It may be administered in simple syrup.

Aspirin may be given in place of the above. The dose is the same as salicylate of soda. It is soluble in about 100 parts of water.

The salicyl derivatives should not be discontinued as soon as the pain stops, but kept up in smaller doses for several days or a week, or longer if necessary. If they are stopped too soon pain and other rheumatic symptoms may return. It is well to give these patients treatment for one or two weeks at a time for a period covering a number of months.

The salicyl derivatives may only act as irritants to the stomach in some cases, and where this happens they must be abandoned and alkalis given instead. It is well to use Vichy water in all cases. In addition to this bicarbonate of soda or the acetate or citrate of potassium may be given. The latter is conveniently given with syrup of lemon and water. Sufficient should be given to render the urine alkaline, and then the dose reduced so that the urine is kept slightly alkaline. From 0.06 to 0.3 gm. (1 to 5 gr.) of any of the above at a dose may be used, or more if necessary. When salicyl derivatives can be given the pain can generally be controlled, but when they are not well borne, opium, in the form of Dover's power, or morphine in small doses may be given to control it.

The after-treatment of rheumatism is very important. Iron for the anemia and small doses of quinine or strychnine are very useful in building up the little patients. Cod-liver oil is of great service. Such children require a large amount of fatty food. An out-of-door life in a dry climate benefits many patients.

**Chronic Fibrous Rheumatism.**—This is a rare disease in children. It has been described by Jaccoud and others. The lesion consists of a thickening of the tissues about the joint and of the joint capsule itself. It may develop after several attacks of acute rheumatism, and is said sometimes to come on insidiously without any very acute symptoms

ever being noticed. Several joints are, as a rule, affected. There may be endocarditis or pericarditis, and rheumatic nodules have been noted in some cases.

**Diagnosis.**—This is made on the repeated attacks of rheumatism, the heart, or other lesions, and the nodules when present.

**Prognosis.**—Prognosis as regards life is good, but as regards cure is bad, as the joints are apt to remain uninfluenced by treatment.

**Treatment.**—Jaccoud claims to have gotten good results from the use of the salicyl derivatives. They are not adapted for continuous use, however. Iodide of potassium may be tried, and guaiacum has been recommended. If there is pain the administration of methyl salicylate 0.18 c.c. (3  $\text{m}$ .) with colchicin, 0.00025 gm. ( $\frac{1}{4000}$  gr.), given in a globule several times a day, may be used. Local treatment is probably more effective in giving relief. "Firing" with the Paquelin cantery, as mentioned on p. 580, or any mild form of counterirritation, may be used. Between the attacks of pain, massage and passive movements do much to prevent ankylosis and stiffness of the joint. The use of hot air at high temperatures may be tried. If possible the patient should spend the winter or damp months in a dry, equable climate. Visits to hot springs sometimes give relief. Good food, tonic treatment, and a quiet out-of-door life are the best things when the patient's means allow them.

#### ARTHRITIS DEFORMANS.

Arthritis Deformans, or, as it is called by some authorities, Rheumatoid Arthritis, is not very common in childhood, but it is sometimes met with. Many cases are mistakenly called rheumatism. In 11 out of 92 cases reported by McCrae, the disease began before ten years of age. The etiology is obscure. In some there is a distinct family history of joint troubles.

**Pathology.**—This is not fully understood. There seem to be two classes of cases. In one there is an hypertrophy of the bone with the formation of exostoses and considerable new bony tissue, the joints looking as if liquid bone had been poured on them and allowed to harden. In the second class there is an atrophy of the bones and of the tissues about the joints. In both there is likely to be marked deformity. Later in either case there may be thinning of the cartilages and degenerative changes in the joint, leading to more or less complete ankylosis. In the first class of cases there may be immobility due to the abnormal deposits of bone "soldering," as it were, the bones together.

**Symptomatology.**—Clinically, the cases may be grouped roughly under two heads: First, those where there are acute attacks of arthritis with remissions, during which the joint is apparently normal, each attack leaving the joint more disabled, however, until finally it may be almost or entirely useless. Secondly, those where there is a gradual onset with progressive joint changes. There is sooner or later atrophy of the muscles and deformity.

In the acute attacks there is swelling of the joint with some pain and tenderness, little or no temperature, and a rapid pulse rate.

In all cases there is usually involvement of the lymph nodes. The spleen was enlarged in only 4 of 30 cases studied by McCrae (including adults and children). In the acute form in children known as Still's disease the spleen is enlarged. Nodules similar to the rheumatic ones or identical with them are found in some cases.

**Diagnosis.**—This can be made if one bears in mind the features of the disease. From acute articular rheumatism the attacks differ in the following points: There is much swelling with comparatively little pain and tenderness; the swelling and symptoms do not disappear rapidly as in rheumatism; there is little or no tendency to move from joint to joint; there is an absence of other rheumatic symptoms; there is little or no temperature, but, as McCrae has pointed out, a high pulse rate. Later on or after several attacks, there is deformity of the joint; atrophy of the muscles and sometimes increased reflexes. The heart is not involved, as a rule.

**Prognosis.**—Prognosis, as far as complete recovery is concerned, is bad. The general health is apt to be poor on account of the patient being more or less crippled.

**Treatment.**—Treatment of the acute attacks consists in rest in bed, flannel clothing, regulation of diet, as much as in the acute attack of rheumatism, and attention to the bowels and general condition. "Firing" the joint, baking with hot air, or other mild counterirritation may be tried for the pain. If the pain is very severe, antipyrin or phenacetin, combined with small doses of codein, gives relief. Koplik states that iodide of potassium is the only drug which relieves the pain. Luff<sup>1</sup> regards guaiacol carbonate as a valuable adjunct to the iodide. For gradual forms and between the attacks, a good, nutritious diet, with plenty of milk, eggs, and meat; out-of-door life, massage, and x-rays are recommended. In both classes of cases patient supervision is essential. Massage and x-rays without oversight and direction will not do any good, while with intelligent direction they may bring about great improvement.

**Spondylitis Deformans.**—This is sometimes seen in children. Usually not under thirteen years, however, although it has been noted earlier. It is a form of osteoarthritis affecting the spinal column, hips, and shoulders. It leads to gradual stiffening of the back and the affected joints. Pressure on the nerves may give rise to pain and atrophy of the muscles. It is likely to be mistaken for Pott's disease or for rheumatism. Tuberculin is useful in excluding tuberculosis. The cases seen early and immobilized in plaster casts may be arrested, otherwise the disease goes on to produce absolute immobility of the spine and of the proximal joints. (See Ruhrah, *American Journal of Medical Sciences*, November, 1903.)

**Treatment.** What has been said of the treatment of arthritis deformans applies equally well to this form of that disease.

<sup>1</sup> Clinical Journal, October 7 and 14, 1903.

**Still's Chronic Joint Disease.**—This is a curious form of arthritis deformans peculiar to childhood, consisting of chronic progressive enlargement of the joints, associated with enlargement of the lymph nodes and of the spleen. Garrod believes it to be entirely distinct from the recognized arthritis deformans. It usually comes on before the second dentition, and it is more frequent in girls than in boys. The onset is generally insidious, but may be acute. No heart changes are recorded. Luff<sup>1</sup> gives four important points in its diagnosis from arthritis deformans: (1) enlargement of the lymph nodes; (2) enlargement of the spleen; (3) the peculiar appearance and doughy feel of the joints without grating or bony outgrowths, and (4) the involvement of the knees or wrists with the fingers secondarily affected. Treatment is limited to diet and hygiene.

**Symptomatology.**—The enlarged joints both feel and look as if there was a thickening of the tissues about the joint rather than of the bones themselves, and is smooth and fusiform, without any bony irregularities of the rheumatoid arthritis of adults. There is no grating, although there may be creaking. There is neither redness nor tenderness, except in acute cases, but there may be pain on motion. There is practically always limitation of movement, chiefly of extension. The child may be completely bedridden on account of this. The joints enlarged are usually the knees, wrists, cervical spine, and subsequently the ankles, elbows, and fingers.

The lymph nodes are hard, but there is neither tenderness nor any tendency to break down.

The spleen is somewhat enlarged in most cases; generally it reaches one or two finger breadths below the edge of the ribs. Both spleen and lymph nodes get larger as the joints become worse. If the disease begins before the second dentition there is usually some arrest of the physical development of the child. The mentality is not affected.

**Prognosis.**—Prognosis as far as life is concerned is good; the cases that have died are from some complicating disease. The outlook as regards the disease itself is bad. The cases, as a rule, have a tendency to get worse instead of better.

**Treatment.**—Treatment is the same as for other chronic joint affections along general lines. There is no specific treatment known as yet.

<sup>1</sup> Clinical Journal, October 7 and 14, 1906.



## SECTION VII.

### DISEASES OF THE RESPIRATORY TRACT.

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#### CHAPTER XXIII.

##### DISEASES OF THE NOSE—NASOPHARYNX—LARYNX.

###### ACUTE NASAL CATARRH.

This complaint, the familiar "cold in the head," is even more readily induced in children than in those of more advanced years.

**Ecology.**—Nasal catarrh occurs as a symptom of many of the exanthemata; when primary, it is almost certainly of bacterial origin, and it is more than probable that many varieties of organism are capable of causing it. The nasal mucous membrane must possess a remarkable power of dealing with the numerous bacteria which are constantly entering with the air stream, since the nasal cavities in health, when once the external orifice is passed, are practically sterile. When nasal catarrh is present, on the other hand, cultures from the mucous surface show a large growth of colonies, consisting, as a rule, of one organism in abundance, probably the causative agent in that particular case, and a few colonies of other varieties besides. Acute nasal catarrh is "caught" in two separate ways: First, it may be set up by a lowering of natural resistance through exposure to cold or damp, by violent purgation in enteritis, etc., the organism being already present; or, secondly, it may be handed on by personal contact, so that it becomes a "household cold." In this case the organism probably acquires an enhanced virulence, and individual immunity is readily overcome.

**Symptomatology.**—"Colds" vary somewhat in the prominence of their symptoms. When severe the sufferer may experience some chilliness, with headache and malaise at the onset. The nasal mucous membrane is at first turgid and dry, but soon a watery secretion appears, and the swelling increases, so that nasal respiration becomes difficult or even impossible. This obstruction is most marked in the recumbent posture, causing much discomfort at night, while in infants attacks of suffocation may result and breast feeding be greatly interfered with. After a time

the watery secretion increases and at the end of a day or two becomes considerable in quantity, the swelling of the mucous membrane at the same time lessening, so that nose breathing becomes easier. The discharge is thicker and less in quantity as time goes on, passing through the stages of serum, seromucous, mucopus, and finally drying up altogether after a period varying from one to two weeks. The child is languid during the attack and unfit for any exertion, but brightens up somewhat after the first two or three days are past. In most cases the catarrh is preceded by some amount of pharyngitis; occasionally it starts in the nasopharynx, especially when adenoids are present, and spreads thence to both nose and pharynx. In either case there is a great tendency for the process to pass downward to the lower respiratory passages, and this constitutes the chief danger of nasal catarrh in childhood.

**Treatment.**—Recurring "colds" in children must not be treated lightly; they may lead to bronchitis and bronchopneumonia. A cause must be carefully sought in ill-ventilated or overheated nurseries, in digestive disorders, in injudicious exposure during bathing, or in cold extremities from insufficient covering, and the cause removed before local and general treatment will prove of service.

At the onset of catarrh a hot bath may be given with a hot lemon drink containing some such diaphoretic as spirits of nitrous ether, 0.60 c.c. (m℥x) for a child of one year, or acetate of ammonia, 0.125 c.c. (m℥xx) of the liquor, and the child put to bed, or, at any rate, confined to one room. If the pharynx is congested it may be sprayed three or four times daily with an antiseptic solution, such as izal<sup>1</sup> ( $\frac{1}{2}$  to 1 per cent. sol.) or listerine (1 in 8), or an older child may use a weak solution as a gargle. In infants a few drops of a 1:1000 solution of adrenalin chloride or a solution of cocaine (2 per cent. in water or 1 per cent. in liquid paraffin or alboline) may be instilled into the nostrils immediately before suckling to clear the nose by constricting the swollen membrane. After the "dry" stage of the cold is passed, quinine should be given in tonic doses of from 0.065 to 0.13 gm. (1 to 2 gr.), and if this causes headache, and more especially if the throat continues sore, salicylate of soda may be given at the same time, 0.2 to 0.3 gm. (3 to 5 gr.) for every grain of quinine prescribed.

These drugs are best given in separate mixtures, as they are not readily combined. Quinine may sometimes be given at the onset with advantage, and has some reputation as a means of cutting short the attack. The association of the acute form of nasal catarrh with gastric and intestinal disturbances must be remembered. Soda in the form of the bicarbonate is often of value in this class of cases and a laxative is helpful. The child should be kept indoors for the first few days, especially if there is any liability to bronchitis, but when the secretion becomes mucopurulent he may generally begin to go about if the weather is fine and gradually resume his ordinary methods of life.

<sup>1</sup> Izal is a by-product in coke making. It is insoluble in water, but forms with it a fine emulsion. It possesses powerful germicidal action.

**CHRONIC NASAL CATARRH.**

**Etiology.**—In infants this is commonly due to congenital syphilis, but may be caused by congenital adenoid growths. In older children it is generally associated with the presence of adenoids in the nasopharynx; more rarely polypi, whether mucous or fibrous, may be the cause, or the later syphilitic lesions with gummata and ulceration may be present. A foreign body, as a button, is sometimes responsible for a unilateral discharge in young children. An acute nasal catarrh may be followed by a period of discharge that may simulate the chronic form due to other causes.

**Symptomatology.**—The symptoms consist of nasal discharge with the resulting snuffling and sniffing, and more or less obstruction to nasal respiration. This last may be due to the adenoid growths so often underlying the condition rather than to the condition itself. The discharge is generally mucopurulent, or even purulent, and readily causes excoriation of the upper lip; if due to a foreign body it is unilateral, and often blood-stained. In cases of late syphilitic ulceration, or in rare cases of atrophic rhinitis, it may be of very offensive odor. The condition of the mucous membrane varies greatly in different cases; it commonly appears red and sticky, and may be hypertrophied, so that the passage is occluded, or in rare cases atrophied and coated with crusts. In late syphilitic cases the nasal bones often fall in, giving the familiar saddle-back deformity, or perforation of the hard palate may occur.

**Treatment.**—The cause must be removed, whether this be adenoid growths, polypi, or a foreign body. The two latter are reached from the anterior nares, the mucous membranes being first sprayed or painted with a 2 to 5 per cent. solution of cocaine, which acts both as a constrictant and as a local anesthetic. Polypi are removed with the snare, or a foreign body coaxed out with a hairpin, or grasped with a fine forceps. In cases of hypertrophic rhinitis the swollen mucous membrane must be cauterized, an alkaline or mild astringent douche ( $\frac{1}{2}$  per cent. solution of alum) being used in the intervals. For atrophic rhinitis an alkaline, antiseptic spray is necessary to loosen the crusts, such as borax and sodium bicarbonate, 0.3 gm. (5 gr.) of each to 30 c.c. (1 oz.), the cavities being afterward lubricated and made clean by a spray of from 2 to 10 per cent. menthol in liquid paraffin or alboline.

**DISEASES OF THE NASOPHARYNX.****ADENOID GROWTHS.**

**Etiology.**—The pharyngeal tonsil lies in the roof of the pharynx, extending somewhat on to its posterior wall. It consists of a mass of lymphoid tissue, as does the faucial tonsil, and is, in truth, but a lymphatic gland of peculiar relationship. Its overgrowth constitutes the

disease known as "adenoids," the main symptoms of which are due to obstruction of the posterior nares. In a certain proportion of cases, 30 per cent. according to some writers, this overgrowth is associated with a similar condition of the faucial tonsils. When, on the other hand, enlargement of the tonsils is found in children adenoid growths are present in addition in 90 per cent. of the cases. The growths generally have a wide attachment, and project as vegetations corresponding to the divisions of the gland. They are either soft, when they bleed very readily, or firm from fibrous overgrowth. This difference bears no relation to age, firm adenoids being often found in the youngest subjects.

Adenoids are found at all ages, and are not infrequently congenital, a fact which is too commonly overlooked. An hereditary tendency appears to be present in some cases.

**Symptomatology.** *In Infants.*—The symptoms may appear at birth or be noticed soon after. The infant snuffles loudly, and often there is

considerable nasal discharge; the nasal obstruction may interfere with suckling, and the children are liable to attacks of suffocation during sleep. Occasionally they are brought to the physician for the treatment of reflex nervous phenomena, such as convulsions, laryngismus, or vomiting. These may be associated with growths too small to cause obstruction, or may occur in cases presenting the gross symptoms described above. The typical adenoid facies is not seen in young infants, but the signs are usually sufficiently definite to form the basis for a diagnosis. Snuffling is very noticeable and is due to the presence of abundant secretion; the nostrils are round



FIG. 129  
Usual expression of a boy with adenoids.

and dilated, not flattened laterally, as generally occurs in older children. On examining the throat the tonsils may be somewhat enlarged, but in infancy the adenoids cannot always be reached by the finger, owing to the smallness of the nasopharynx. In cases where the growths are small the symptoms only appear at such times as the obstruction is increased by acute catarrh.

*In Older Children.*—The symptoms often first appear after some illness associated with catarrh, such as scarlet fever, measles, or whooping-cough. The attention may be drawn to the condition either by certain ear complications or by the nasal obstruction itself, and many cases are first seen on account of recurrent attacks of bronchitis and nasal catarrh.



In a marked case the appearance of the child is very typical; the mouth is open and the jaw dropped, giving a vacant expression to the face (Fig. 129); the lips and tongue are dry, the eye is watery and injected, and the nostrils may be mere slits, the alæ being flat and expressionless. Nasal discharge is generally obvious and may cause excoriation of the upper lip.

The child snores at night and may wake half-suffocated at intervals, often with night-terrors, the voice is nasal and toneless, headache is common and mental processes dulled, taste and smell are deficient, and in some cases the child has difficulty in swallowing and, when young, regurgitates his food. Incontinence of urine is sometimes present.

Far complications are common and some deafness the rule. A nasopharyngeal catarrh is constantly present, and attacks of pharyngitis, laryngitis, and bronchitis frequently arise, but apart from these a barking cough may be present as a reflex phenomenon. Habit spasm is observed in some cases.

Deformities appear in connection with the condition; in the face growth is hindered, leading to the narrow jaw with crowded teeth and the high-arched palate. In the chest alteration of shape may arise; pigeon-breast with a deep Harrison's sulcus occurs in some cases, but the most typical deformity is the formation of a hollow at the lower end of the sternum; this may be observed in process of formation, the parts being sucked in with each inspiration.

**Diagnosis.**—This can usually be made from the appearance of the patient, and, on inspecting the pharynx, relaxation of the soft palate, lymphoid masses on the pharyngeal wall, and often enlargement of the tonsils will be noticed. Having decided that some nasal obstruction is present it is better to defer examination of the nasopharynx, especially in a young child, until an anesthetic has been given and the patient prepared for operation; the child is thus spared the distress which a digital examination without an anesthetic occasions. Other conditions which may simulate the obstruction of adenoids are: 1. *Bony obstruction* due to insufficient size of the posterior nares, a low pharyngeal vault, prominence of the crest of the vomer, or a forward projection of the vertebral column. These are liable to occur in ill-nourished and rickety children. 2. Thickening of *soft parts* throughout the nose and over the internal pterygoid plate and tuberosity of the palate. 3. New-growths other than adenoids. 4. Retropharyngeal abscess.

In young children snuffling is generally the most noticeable symptom and suggests the presence of congenital syphilis. The absence of a syphilitic family history, of specific eruption, and of the anemia with yellow skin commonly found in this disease will generally serve to distinguish them. The dilated nostril, so often seen with adenoids in infancy, may be of assistance.

**Treatment.**—When the condition is found in early infancy surgical measures may be delayed if the symptoms of obstruction are lessened by other treatment, and the coincident catarrh is not severe. Operation in early infancy is difficult, and too much must not be prom-

ised as it is likely to be incomplete. The catarrh which increases the obstruction can often be kept in abeyance by instilling a few drops of a 1 per cent. solution of resorcin into the nostrils four or five times daily, and for the nervous phenomena sedatives may be tried. Every plan for building up and strengthening the mucous membrane should be used. Operation is, however, sometimes performed with good results as early as the third or fourth month.

In older children it is well to wait until any acute condition, such as bronchitis, is past before an operation is undertaken, but treatment must not be needlessly postponed, owing to the deleterious effect of the adenoids on the general health, the danger of ear complications, and the liability to diphtheria and scarlet fever which the condition of the nasopharynx entails. The patient is prepared for the administration of an anesthetic, chloroform being that most commonly chosen in England and ether in America. When the child is "under" the anesthetic, the head is hung over the end of the operating-table and the adenoids palpated. Enlarged tonsils, if present, are removed with the guillotine. Adenoids are most readily removed with a Gottstein curette. The sharp ring is passed behind the soft palate, and, with a downward movement of the handle, the adenoids are cut from before backward, often coming out as a single mass. The parts are palpated with the left forefinger, and the cutting process repeated till the space is clear. Forceps are preferred by some operators, in which case care should be taken to avoid injury of the entrance to the Eustachian tubes or the nasal septum. Hemorrhage is smart, but quickly ceases. Douching or other after-treatment is best avoided, and blowing the nose should be prohibited for a day or two. Occasional troubles arising as the result of operation are stiff neck, otitis media, and infection with scarlet fever or diphtheria. A white slough on the stump of the tonsil, often present after the operation, must not be mistaken for the membrane formed by the latter disease.

After the adenoids have been removed education in nasal breathing is required, or a child may remain a mouth-breather from mere habit.

#### ACUTE PHARYNGITIS.

The two conditions, Acute and Chronic Pharyngitis, which have been described (p. 192), are briefly mentioned here because of their association with pathological changes in the respiratory passages.

Acute Pharyngitis occurs as a complication of certain of the specific fevers, especially scarlet fever and measles, but is often primary, in which case it very commonly spreads upward to the nasal cavities or downward to the respiratory passages. It results from the growth of various micro-organisms, cold and damp acting as predisposing causes.

**Symptomatology.** Pain on swallowing is the most marked symptom, but in young children the condition often gives rise to noticeable constitutional disturbance. There may be vomiting at the onset and chilli-

ness; the temperature rises, sometimes to a high level; there is constipation, and the child seems out of sorts. The pharyngeal mucous membrane appears red and somewhat swollen, the tonsils often sharing in the inflammation. After a day or two the symptoms subside or disappear after spreading in many cases to the nose or respiratory passages.

**Treatment.** The child should be put to bed while the fever lasts, the bowels moved with a calomel purge, and a cold compress applied to the throat. The diet should be fluid, and cold water should be given freely to allay the sensation of dryness and thirst. In addition, salicylate of soda, 0.13 to 0.18 gm. (2 or 3 gr.), for a child of three years, with quinine, 0.05 gm. (gr.  $\frac{1}{2}$ ), or phenacetin, 0.13 gm. (gr. 2), may be given every four hours, the throat being sprayed with listerine (1 in 8) or with 10 per cent. menthol in liquid paraffin from an oil atomizer.

**Chronic Pharyngitis.**—This is generally associated with an increase of lymphoid tissue in the pharyngeal wall, a condition which commonly accompanies the presence of adenoid growths in the nasopharynx, and may be congenital, or may result from attacks of acute catarrh, especially after scarlatina or measles. The disease gives rise to a loud, hard cough, and, in addition, the symptoms of adenoids with which it is commonly associated may be present. On examination the pharynx appears congested, with pale cushions of lymphoid tissue projecting from its wall.

**Treatment.**—This consists in improving the general health, removing the adenoid growths when present, and painting the mucous membrane with astringent applications, such as equal parts of liquor ferri perchloridi and glycerin. These methods failing, the lymphoid tissue in the posterior pharyngeal wall should, in older children, be destroyed with the galvanocautery.

#### FOLLICULAR TONSILLITIS.

This disease is rare in infancy, but not uncommon in older children, especially where chronic enlargement of the tonsils is present. In infancy it may be associated with an acute influenza or a disturbance of digestion. The subjects of acute rheumatism are not infrequently attacked, in which case the condition may be complicated by acute endocarditis. Children who are below par and who are allowed sweets and pastry are liable to attacks of tonsillitis more often than children who are given proper food.

**Symptomatology.**—The symptoms are severe and of sudden onset. Vomiting may occur, though this is a more usual feature in cases of diphtheria. Chilliness is common, or even a rigor, and headache and pains in the back and limbs are generally complained of. The temperature rises rapidly to a high level, 102° or 103° F. being common, and the bowels are confined. The local symptoms are often slight. The child appears flushed and feverish, the tongue is coated and foul, and the lymph nodes in the neck generally enlarged. Both tonsils are affected and are swollen and reddened, with plugs of yellowish secretion protruding from the crypts. The symptoms last but a few days, and the



exudate rapidly clears, though some enlargement of the tonsils may remain for a week or longer.

**Diagnosis.**—Changes in the throat are frequent in scarlet fever, and a diagnosis from the ordinary follicular tonsillitis may be difficult unless the exanthem is looked for. Diphtheria with membrane may be mistaken for tonsillitis, but in the latter disease the symptoms of fever, headache, muscular pains, and lassitude are more decided, and are described under the article on Diphtheria (p. 385).

**Treatment.**—The child should be put to bed, and 0.065 gm. (1 gr.) of calomel administered. Quinine and sodium salicylate are useful in the later stage of tonsillitis, and especially so where there is a rheumatic diathesis. Phenacetin, 1.3 gm. (2 gr.) for a child of two or three years, may be given. In young children local treatment may be dispensed with, but in older children an antiseptic gargle or spray, such as Dobell's solution, Seiler's antiseptic throat tablets in solution, or listerine (1 in 8) should be used every three or four hours.

#### CIRCUMTONSILLAR ABSCESS.

Circumtonsillar Abscess, or Quinsy, is uncommon in childhood. When it occurs the constitutional symptoms are similar to those of follicular tonsillitis, though often less severe, while, in addition, marked local symptoms are present, namely, great pain on swallowing and difficulty in unclosing the jaws. The condition is always unilateral, and the inflammation often starts behind the tonsil, so that this organ appears thrust forward into the mouth.

**Treatment.** The bowels should be well opened with a calomel purge and quinine and salicylate of soda given. Salol is recommended by many. The diet must be fluid, but ample, and ice-cream and custards may be of advantage when fluids are difficult to swallow. Alcohol may be given with advantage where suppuration seems inevitable. When pus has formed it must be evacuated by means of a guarded bistoury.

#### CHRONIC HYPERTROPHY OF THE TONSILS.

This is generally associated in children with enlargement of the nasopharyngeal tonsil or adenoids. The hypertrophy is in some cases congenital, but generally it arises from repeated attacks of tonsillitis or follows catarrhal conditions, influenza, or some one of the specific fevers—diphtheria, scarlet fever, or measles. The symptoms are commonly associated with those more important ones due to adenoid growths, but when these are eliminated there remain the "throaty" voice, as if the mouth were full of food, dry cough in some cases, and the tendency to repeated attacks of acute inflammation.

**Treatment.** This consists in the removal of the tonsils with a guillotine, or, if operation is objected to by the parents, their gradual reduc-



tion with the galvanocautery. Better results are observed when the child is in the open air and sunshine, and as the condition is so often associated with a debilitated state of the system, hygiene should not be overlooked. Cod-liver oil and iron are useful in many cases.

### RETROPHARYNGEAL ABSCESS.

Acute Retropharyngeal Abscess is of not uncommon occurrence in childhood; tuberculous abscess, on the other hand, is rare.

**Acute Retropharyngeal Abscess. Etiology.**—This disease occurs in infants below the age of two years, and is generally secondary to a pharyngeal or nasopharyngeal catarrh. It arises from infection of the lymph nodes lying on either side behind the pharyngeal wall.

In three cases in which I examined the pus aspirated immediately before operation, one contained the pneumococcus, one a streptococcus, and the third an organism of doubtful identity; in addition, all grew a few colonies of a common mouth organism, a large diplococcus of gonococcus-like shape.

**Symptomatology.**—The symptom which first calls attention to the condition varies in different cases; it may be dyspnea, or swelling of the lymph nodes of the neck, or nasal discharge, accompanied by snoring during sleep. The dyspnea is mainly inspiratory, but may be double, and is worse in the recumbent posture; it not infrequently causes some recession at the base of the chest, which is more marked when the child is excited or disturbed. The breathing is rattling, and snoring or stertorous; there is hard cough, which may be paroxysmal, and the voice is nasal, but generally clear. The child appears ill, and the temperature is raised, often to 101° or 102° F. Dysphagia is a common symptom, especially if the swelling is low down in the pharynx. The mouth is generally open, the head inclined to one side to relax the muscles, or sometimes retracted. The open mouth and evident distress are well shown in Fig. 130. The neck is stiff and may appear generally enlarged, or there is a swelling below one ear due often to a secondary lymph-node infection, but sometimes to the abscess itself, which may appear as a fluctuating swelling in the side of the neck in front of the sternomastoid muscle, and may extend, in some cases, from the angle of the jaw nearly to the clavicle. On inspection and palpation of the pharynx a tense, globular swelling is found on one side of the pharyngeal wall. To the touch it is elastic in the early stages, but later fluctuation is detected. The pharyngeal mucous membrane is generally inflamed and often covered with mucus, and it may feel "boggy" to the examining finger.

**Course.**—After prompt surgical treatment the general condition rapidly improves and the abscess heals in from one to two weeks, the mortality for such cases being very small. When neglected, or in bad cases following scarlet fever, measles, and erysipelas, the abscess may track down the neck into the mediastinum, and the child die with septic bronchopneumonia or occasionally with empyema.

**Treatment.**—The abscess must be promptly opened as soon as found. This is best done through the pharyngeal wall, either with a guarded bistoury or by means of a director, the head being held forward so that the pus, generally but a few drachms in quantity, runs into the mouth. Gentle pressure at the sides of the neck may help to evacuate it. Afterward an antiseptic douche or spray, such as 3 per cent. resorcin, or izal 1 in 300 to 500, may be used for a few days. In cases where a large fluctuating swelling appears in the neck, especially when a septic source for it can be traced, it is best opened and drained externally. Some surgeons prefer this latter method, no matter whether the abscess be submucous or subcutaneous, as they believe it essential to clean out the

FIG. 130



Cotter's case of retropharyngeal abscess before operation.

broken-down lymph nodes. The nature of the operation must be determined by the character of the case, but as there is immediate relief from a simple incision through the pharyngeal wall, this operation can be done without delay.

**Tuberculous Retropharyngeal Abscess.**—These cases are rare and occur in older children. They can generally be diagnosed from an abscess by their painless character, their slow development, the age of the patient, and the fact that often the swelling is less localized to the lateral pharyngeal region. They arise from caseation of the postpharyngeal lymph nodes, or, in some cases, are due to spinal caries, in which case the symptoms of that disease will be superadded.

**Treatment.**—These abscesses must never be opened from the pharynx, on account of the danger of sepsis. As a rule, either the abscess itself or some adjoining tuberculous nodes will be found in the side of the neck, and an operation must be performed at this point, an incision being made along the anterior margin of the sternocleidomastoid, the nodes removed, and the abscess drained.

## DISEASES OF THE LARYNX.

### ACUTE LARYNGITIS.

Mild forms of this disease occur as the result of the inspiration of cold or damp air, and may exist alone, or in combination with nasal and pharyngeal catarrh, or with tracheobronchitis. The voice is hoarse or may be lost, and a "croupy" cough is present, but the condition causes little or no general disturbance. More serious aspects of the disease are best described under the term "laryngitis stridulosa," of which slight and severe varieties exist. In addition, edematous laryngitis claims a few words of description.

**Laryngitis Stridulosa.**—The slight attacks are commonly termed *false croup*, or *spasmodic croup*, and have generally been ascribed to a spasm of the glottis, but there is at least as much evidence against as in favor of this theory. Their resemblance to the more severe form of laryngitis about to be described seems to me so much closer than to that truly spasmodic affection laryngismus stridulus, that I have grouped the two together under the heading of Laryngitis Stridulosa. The most probable explanation of the attacks is a swelling of the laryngeal mucous membrane, comparable to that which so commonly obstructs the nasal fossæ when the recumbent posture is assumed at the onset of a nasal catarrh.

**Etiology.**—The disease attacks children during the first dentition, one and a half or two years being a common age. The condition very commonly occurs at the onset of measles, in some cases before any symptoms of that disease have appeared, or it follows in the wake of measles, whooping-cough, or influenza.

**Symptomatology.**—The onset may be unexpected, or it may be preceded by a nasal catarrh, slight cough for a day or two, or in some cases a slight laryngitis has been present for a few days before the urgent symptoms arise. As a rule, the attack commences in the night; the onset is sudden, with the peculiar, loud, brassy, or "croupy" cough, and the breathing becomes stridulous and difficult. The symptoms increase until in many cases the distress is urgent, and each inspiration is accompanied by recession at the base of the chest, the end of the sternum, and in the supraclavicular and suprasternal fossæ. The child appears anxious and very restless, the skin is flushed and sweats freely, and some cyanosis is evident. The temperature is often but little raised, and when high this generally depends on some



accompanying pharyngitis. The pulse is increased in frequency, but the pulse-respiration ratio is not disturbed. The voice is generally hoarse and metallic, but may be natural, and stridor is usually inspiratory only, but may accompany both inspiration and expiration. On examination the tongue is found coated, and the fauces and tonsils nearly always reddened; nasal catarrh can generally be observed, and, in cases preceding measles, conjunctivitis also. The lungs may show a few bronchitic rales, but often the laryngeal stridor is the only sound audible. The urgent symptoms generally last from one to three days and are subject to paroxysmal exacerbations, especially at night. When they have passed off some amount of laryngitis remains for a week or longer. The attack may be so severe as to necessitate tracheotomy, but this is uncommon. As a rule, the dyspnea becomes gradually less, the stridor disappears during sleep and only returns with deep or hurried respiration; finally it goes altogether, leaving hoarseness and cough for a variable period.

**Diagnosis.**—The diagnosis between laryngeal obstruction due to simple catarrh and that due to diphtheria is often very difficult until the result of a culture is obtained. Moreover, membranous laryngitis may occur apart from diphtheria, and when membrane is discovered on the tonsils in a case of laryngeal stenosis, this may be due to the action of streptococci or other organisms. If the palate is invaded it is almost certainly diphtheritic. A culture from the throat should be taken in all cases, and if membrane is present films from this should be immediately examined if possible. In stridulous laryngitis the onset of the obstruction is usually sudden, involving inspiration only; the dyspnea tends to be paroxysmal, the voice is loud and hoarse, but may be clear, and the cough ringing and brassy. In laryngeal diphtheria, on the other hand, the stenosis is of more gradual development, but is progressive, involving first inspiration only, but later both inspiration and expiration; the voice is muffled or absent. The effect of treatment is often of valuable assistance, many of the catarrhal cases improving rapidly in the moist atmosphere of the steam tent, while this has no effect on the obstruction of diphtheria. The temperature may be of some aid, since if it is high, simple laryngitis is more probable; whereas, if a low temperature is present, with a well-marked throat inflammation, this is very likely to be diphtheritic. Vomiting is common at the onset of diphtheria, and the child, as a rule, appears more ill and "poisoned" than in simple laryngitis, which is a purely local process. Albuminuria occurs in a proportion of diphtheritic cases, but is not present in simple laryngitis.

**Edematous Laryngitis.**—This occurs in young children as the result of sucking the spout of a boiling kettle, or by drinking boiling hot tea or other liquids, and a number of cases have been recorded. The nature of the condition is readily recognized by the thin white pellicle covering the inside of the mouth and leaving a raw surface when detached. Symptoms of laryngeal stenosis soon supervene and reach their height within twenty-four hours, often rendering tracheotomy necessary.



**Treatment.**—In cases of even slight laryngitis the child should be kept indoors. The room should be warm and the air moist, drinking-water should be freely given, and an alkaline mixture containing ipecacuanha prescribed for the purpose of loosening secretion.

B—Vinum ipecacuanhæ . . . . .	0.30 c.c.	(℥v).
Sodii bicarbonatis . . . . .	0.09 gm.	(gr. iss).
Sodii chloridi . . . . .	0.03 gm.	(gr. ss).
Aque chloroformi . . . . .	q. s. ad 4.00 c.c.	(3j).

This may be repeated every three or four hours for a child of one year.

For cases of so-called *spasmodic croup*, emetic doses of ipecacuanha should be given in 0.6 gm. (gr. x) of the powdered root, or 4 c.c. (1 drachm) of the vinum every one-fourth hour till vomiting is induced. If no vomiting follows these large doses of the drug, no harm but often benefit accrues from them. At the same time the air around the child must be moistened by means of a steam kettle, warm drinks given, and flannel or spongiopiline wrung out of hot water placed round the throat.

In cases of *serious laryngeal obstruction*, if any doubt exists as to the simple or diphtheritic nature of the condition, it is well to administer 4000 units of antitoxin at once. Subsequently a calomel purge should be given, and the treatment mentioned above pursued, namely, a steam tent, hot fomentations, and emetic doses of ipecacuanha. In addition, the inhalation of compound tincture of benzoin from the surface of hot water may be of assistance. After vomiting has occurred the secretion must be kept loose with doses of vinum ipecacuanhæ, 0.3 to 0.6 c.c. (℥v to x), or vinum antimonialis, 0.3 c.c. (℥v), in a diaphoretic mixture containing ammonium acetate, 1.3 c.c. (℥xx) of the liquor, every three or four hours. The child must remain in a warm room until the catarrh has subsided, and the normal conditions of life must be resumed with caution. In some cases intubation or tracheotomy becomes necessary in spite of treatment, but if the case is believed to be simple laryngitis and the medical attendant can remain on the spot, operative measures should be postponed as long as possible.

The treatment of *edema of the larynx* to be successful must be energetic, and that which most commends itself is the administration of calomel after the manner originally recommended by Bevan. The drug is given as soon as a case of the nature described comes under observation, even before laryngitis arises, 0.06 gm. (1 gr.) being administered every half-hour till green stools are passed. At the same time cold compresses must be applied to the throat, and a laryngeal spray of picric acid has been recommended. If, in spite of treatment, the stenosis becomes extreme, tracheotomy must be performed.

#### CHRONIC LARYNGITIS.

This is rare in children, the symptoms being hoarseness and, generally, some amount of cough. It is commonly the outcome of a protracted

acute catarrh, or, in infants, is of syphilitic origin. The former cases occur especially where adenoids are present, and the removal of these generally cures the disease; in addition, the use of warm applications to the throat and the internal administration of potassium iodide in an alkaline mixture may be of benefit, or, failing these, change of air and tonic remedies should be tried. Syphilitic laryngitis is not very uncommon in infancy, and its treatment is that of the congenital disease it accompanies.

### LARYNGISMUS STRIDULUS.

This disease is a pure neurosis, depending for its symptoms on attacks of spasm, generally limited to the glottis, but in some cases passing on to other areas, so that a partial or general convulsive attack ensues. It occurs in infants between the ages of six months and two years of age, is most common in male children, especially so in those suffering from rickets, and is often associated with the presence of adenoid growths. The condition appears to be most prevalent in the cold months of the year, possibly because the obstruction of adenoids is more marked at these times.

**Symptomatology.**—The spasm is generally brought on by excitement, or by an attack of crying or coughing. It may lead only to slight inspiratory difficulty, giving rise to a crowing sound, resembling the cry of a seagull, accompanying several inspirations. In the more severe attacks the head is thrown back, the face becomes livid, and after a long moment of silence the breath is at last drawn in again with a loud, crowing sound. Such an attack may end in loss of consciousness, or even death, in which case no sound is uttered. In other cases a general convulsive seizure occurs, or the attacks alternate with general convulsions. Carpopedal spasm is present in some cases and persists between the attacks. The attacks may be few and far between, or twenty or thirty may occur daily. The tendency to them lasts from a few days up to many weeks or months.

**Treatment.**—This is directed toward improving the general nutrition of the child, especially in relation to the presence of rickets, in lowering the nervous susceptibility, and in removing or quieting any local predisposing cause, such as adenoids, that may be present. For the first an ample proteid diet, attention to the digestive functions, fresh air and exercise, cold bathing, and such tonics as cod-liver oil and iron or hypophosphites must be given. For the second bromides and chloral are generally considered the most reliable remedies; the former may be given in doses of 0.3 gm. (5 gr.) of the sodium salt, the latter in 0.13 gm. (2 gr.) doses to a child of one year, and these may be repeated every three or four hours at first, the intervals being afterward lengthened. If adenoid growths are discovered, these may be removed, or, if the infant is young, the nasal catarrh which accentuates their presence may be kept in abeyance by the use of antiseptics, such as 1 per cent. resorcin, instilled into the nostrils, operation being postponed till a later date. During the attack

itself there is little time for treatment the face may be sprinkled with cold water, but nothing further can be done; unless a general convulsive seizure supervene, in which case chloroform may be administered.

#### CONGENITAL INFANTILE STRIDOR.

**Pathology.**—Various explanations of this ailment, both functional and structural, have been advanced. Dr. D. B. Lees, in a case fatal from diphtheria, found the epiglottis folded on itself, and the arytenoepiglottic folds in contact, so that the upper aperture of the larynx was greatly narrowed. These appearances have been confirmed by Dr. G. A. Sutherland and Dr. Lambert Lack by laryngoscopic examination during life, the thin, flaccid folds bounding the aperture being observed to fall together during inspiration and again separate with expiration. The structural change persists with growth, but the increasing rigidity of the surrounding parts serves more efficiently to keep the aperture patent.

**Symptomatology.**—The symptoms are present from birth and consist of an inspiratory croak, sometimes likened by the mother to the clucking of a hen. In some cases the croaking sound is audible during expiration, as well as inspiration. It is absent during quiet breathing, as in sleep, but reappears when the respiration deepens on any excitement, or with crying, coughing, and sometimes in feeding. The cry is natural, and no signs of respiratory obstruction are present during the quiet intervals; but when the stridor is present some inspiratory recession of the unprotected parts of the chest can be seen, and occasionally the alæ nasi work. In marked cases some amount of permanent chest deformity may be set up. The stridor remains the same or increases up to the age of nine to twelve months, after which it gradually becomes less marked, and ceases at eighteen months to two years of age, but will often reappear with unusual respiratory efforts for some years later.

**Treatment.** The condition seldom leads to serious trouble, and from its nature is outside the bounds of drug treatment. The parents should be reassured, and the general condition of the child attended to, more especially with a view to avoiding the risks of a superadded catarrh of the respiratory tract. Tracheotomy must be kept in mind as the only treatment available for rare cases where suffocation seems likely to ensue.

#### NEW-GROWTHS OF THE LARYNX.

The new-growths include *Papilloma*, *Fibroma*, *Myxoma*, *Chondroma*, *Sarcoma*, and *Epithelioma*, of which all but the first are very rare.

The **Papillomata** are either congenital, or follow one of the exanthemata in children of about five or six years of age. Hoarseness is the first symptom to appear, and this continues for a long time before any laryngeal obstruction is brought about. The latter usually gives rise to violent attacks of dyspnea, in one of which the child may die if no



treatment is adopted. The growths are warty, with a wide base of attachment, or long and branching; they are generally multiple and cover the mucous membrane between the epiglottis and just below the vocal cords, often hanging in thick tufts, which fill up the narrow space.

FIG. 121.



Papilloma of the larynx. (Ellerich.)

**Treatment.**—This consists in the performance of tracheotomy, after which the growths separate and come away in the secretions, the process of cure lasting from six months to a year. If they are removed by operation recurrence nearly always occurs. Intubation is not advisable, the tube irritates the tumors and tends to hasten their growth.



## CHAPTER XXIV.

### THE LUNGS IN EARLY CHILDHOOD—BRONCHITIS—PULMONARY COLLAPSE—BRONCHIAL ASTHMA.

#### THE LUNGS IN EARLY CHILDHOOD.

The lungs of the child are both easier and at the same time more difficult of examination than are those of the adult—easier in that the chest wall is thinner and transmits the pulmonary signs with more readiness; more difficult on account of the emotional nature of the child, which often makes detailed examination impossible, and also on account of the greater difficulty of interpretation of the signs discovered. These two factors leading to difficulty are especially present during the first few years of life; in older children examination is generally easy, the signs readily obtained, and their interpretations more nearly that which is required in the case of the adult.

The child's confidence should be gained, if possible, while a history is being obtained and the general shape and movements of the chest observed. Percussion should be lightly performed with one finger only, both because better results are so obtained and because the child is less likely to raise objection; in some cases it is wiser to leave percussion to the last. The lungs of a crying child are not very difficult to examine, but those of a screaming, frightened child are often impossible. The back of the chest generally gives the most important signs of disease, and in a difficult case may be with advantage the part first examined. The child should be held looking over the nurse's shoulder or sitting on a high stool or table. The arms must be drawn forward and the shoulders kept even. A trifling irregularity in position will alter considerably the signs on the two sides, both to percussion and auscultation, in the normal chest of a child.

The child's thorax is more nearly circular than that of the adult, with the result that costal respiration is little efficient and is largely replaced by increased activity of the diaphragm. As a result of the shape of the chest, the lateral region is proportionately large, and must always be examined separately in the child, the arm being raised above the head for that purpose. Not uncommonly the earliest signs of a croupous pneumonia are by this means discovered at the very summit of the axilla. The thoracic walls are soft and yielding in childhood, more especially when rickets are present. For this reason, any respiratory obstruction readily leads to deformity of the chest; moreover, collapse of the lung is very readily produced in parts where the thoracic suction is feeble.

Anatomically, the lungs of the child present certain noticeable differences to those of the adult; the air tubes are of larger area in proportion to the lung tissue, which, perhaps, explains the greater frequency of bronchitis and bronchopneumonia in early years; the interstitial framework of the lung is more noticeable than in adult life, and the alveoli are considerably smaller, leading to the finer granulation of the cut surface in croupous pneumonia.

With regard to the pathological significance of various regions, the apex is of less importance than in adults, since pulmonary tubercle does not often start at this point in infancy; croupous pneumonia of the upper lobes is very common in childhood, and pulmonary collapse occurs at the base, in the thin edges, or as a narrow band along the posterior border near the vertebral bodies.

In childhood the middle portion of the lung has considerable pathological significance, as it is so commonly the seat of collapse or tubercle spreading from the lymph nodes at its root. Localized pleural effusions also may occur over its surface and may simulate conditions of cardiac enlargement. Careful examination of the middle part of the lung must, therefore, never be neglected.

Certain warnings may be advantageously given with regard to the normal pulmonary signs of childhood. In the first place it is usual, especially in thin children, to find bronchovesicular breathing in the interscapular regions behind, and this bronchial quality is, as in adults, more marked and more widely diffused at the right apex than the left. Secondly, in percussing the bases of the chest the liver may give a suspicion of impairment on the right side, while the stomach note is very apt to overpower the dulness of fluid in a small effusion at the left base. Thirdly, loud sounds, such as bronchial breathing and loud friction sounds, are readily transmitted across the chest from one side to the other, so as to appear present at both. They may also be heard over the upper area of the abdomen. Lastly, a cracked-pot sound may often be obtained on percussion over the front of the chest in an infant, particularly when crying.

The thoracic lymph nodes are of great importance in the early years of life, owing to the frequency with which they are the primary focus from which tuberculosis of the lung arises. They cannot be themselves examined during life, but signs of consolidation in the intrascapular region may sometimes point to a spread of tubercle from them, and the presence of enlarged tracheal nodes beneath the manubrium may confirm the diagnosis in a case of bronchopneumonia of doubtful nature.

#### ACUTE BRONCHITIS.

An inflammation of the bronchial tubes is one of the commonest maladies of childhood, and, though often insignificant in itself, it must always be treated with respect, on account of its relationship to more serious diseases. Prompt treatment is always needed, both immediate, to avert the danger of bronchopneumonia and pulmonary collapse, and sub-

quent, to prevent future attacks and the risk of a chronic susceptibility arising.

**Etiology.**—Bronchitis is most common in the cold months of the year. The exciting cause is some variety of micro-organism, differing in nature according to the origin of the infection. The organisms connected with the infective fevers, many of which have bronchitis as an accompaniment, are doubtless causative in this respect—influenza, whooping-cough, measles, and many others—in some of which the organism is recognized, and in some its presence merely assumed. In addition, the primary cases are due to bacteria, probably of more than one species, derived from the nose and pharynx, and any organism causing catarrh of these chambers may also cause catarrh of the bronchial tubes.

The most important of *general predisposing causes* is what may be termed “injudicious coddling.” Perhaps the child has already had an attack of bronchitis and the parents greatly fear a return of the trouble. The result is that a “hot-house” system is instituted whereby the child’s susceptibility is greatly increased by overheated, stuffy rooms, an excess of heavy clothing, causing the skin to remain moist and sweating, and great deficiency of fresh air and exercise. Under these conditions the smallest exposure will cause a fresh catarrh and, thereafter, caution is redoubled with further disastrous results. Such children must be gradually acclimatized to more healthy conditions so that their abnormal susceptibility may be reduced.

In infants two common conditions are often the starting point of bronchitis, namely, *teething* and attacks of *diarrhea*. The former probably acts by increasing susceptibility through the presence of a certain amount of catarrh and pyrexia which seem incidental to the process; in the latter one might well suppose that some absorption of toxins from the intestinal tract was responsible, but the same results may sometimes be observed from free purgation with drugs, and I cannot help attributing it also to a heightening of susceptibility.

The condition commonly known as *chill* is perhaps best described as an “immediate” predisposing cause owing to its close relation to the bronchitic attack. It is induced by general or local change of surface temperature, especially in certain susceptible individuals, and is probably a vasomotor phenomenon. It has been experimentally shown that the application of cold to the skin is followed by a reflex contraction of the tracheal vessels, followed by congestion and an increased flow of mucus. This is the condition which probably forms the starting point of many bronchitic attacks by offering a convenient nidus for local infection.

Of *local predisposing causes*, adenoid growths are the most important, acting, probably, by keeping a growth of organisms always handy on the catarrhal surfaces of the nasopharynx, but also through the unhealthy habit of mouth-breathing, whereby the air enters the passages in an unwarmed and unfiltered condition.

**Pathology.**—The bronchial mucous membranes become swollen and injected, and the secretion, at first diminished in quantity, soon becomes

increased, passing through the stages of serum and mucoserum to mucopus as the disease advances. The mucous membrane only is attacked at first, but if the inflammation continues the whole thickness of the bronchial wall may be involved, leading to a dilatation of its channel. When the smaller tubes are affected, plugging of theirumen readily occurs, leading to the formation of areas of collapse, and these to areas of compensatory emphysema. Collapse is found, especially in the lungs of infants, generally as shallow areas down their posterior surfaces.

**Symptomatology.**—Bronchitis may be divided up for convenience under three headings, though the division must be to a large extent arbitrary.

1. *Tracheobronchitis*, in which the trachea and large tubes are affected, giving rise to cough, but to little or no constitutional disturbance. The process generally spreads down from the pharynx or nasal cavities and, for signs, either a few râles are heard at the root of the lungs or nothing is found on examination.

2. *Bronchitis of the medium tubes*, which comprises the common cases of acute and severe bronchitis with constitutional symptoms.

3. *Capillary bronchitis*, a widespread inflammation of the finest tubes extending into the lobular bronchioles and accompanied, in most cases, by inflammatory changes in the lung alveoli.

In the *slight cases* of bronchitis where only the larger tubes are involved the general health may remain unaffected, and, beyond some cough, no discomfort may occur. In more marked cases the temperature may be somewhat raised, and the cough hard and distressing with some soreness under the sternum.

In *severe cases* of bronchitis the symptoms are often of sudden onset, or sometimes slight cough is noticed for a day or two, and then the condition gets rapidly worse, as at the onset of many cases of bronchopneumonia. Vomiting may be an initial symptom, a hard cough develops, the child is feverish, restless, and refuses food, and the breathing becomes rapid and distressed. Pain under the sternum or in the epigastrium is described by older children, and also headache in many cases.

The child appears flushed and feverish, with a warm, moist, or sweating skin; the conjunctivæ may be injected and watery, and nasal discharge is often noticeable. The breathing is rapid and difficult, the alæ nasi are active, and the child gives vent to a hacking cough at intervals. The tongue is moist and coated, the pharynx injected, the temperature raised, and the pulse rapid and full, perhaps 140 per minute in a child of one year. The attack lasts from five or six days up to ten days or a fortnight in most cases.

The *cough* is dry and hacking at first; later it becomes looser, chokier, and sometimes spasmodic. It is generally painful at the beginning and may cause crying or attacks of screaming in young children. Children do not expectorate their phlegm, but occasionally some is brought up with vomiting, more commonly in the later stages, when it is pur-



lent and more abundant; at the beginning it is thick, viscid, and tenacious.

In young children a *high temperature* is generally found at the beginning, and the height to which this may rise is simple bronchitis is, I think, not always sufficiently realized. A temperature of 102° or 103° F. is not at all uncommonly seen during the first few days—thus a child of ten months had a temperature of 103.8° F. for the first three nights, the attack was finished by the fifth day; another child of two years and four months had a temperature of 103° F. for three nights, with no subsequent rise. These cases showed no signs of teething, but when this accompanies bronchitis the temperature is usually high, often 103° F. or more, as in a child of one year and three months in whom the temperature rose to 105° F. on one occasion. A persistence of temperature after three or four days is significant of further inflammation.

The point about the high fever in bronchitis is its short duration; it rarely lasts more than three or four days and thereafter the temperature is much lower, or normal. Occasionally it remains as high as 101° to 103° F. for a week or even more. The temperature usually shows considerable fluctuations, as in bronchopneumonia, and is very irregular in its course, being high in the evening and low in the early morning hours, though occasionally the inverse type is observed, high in the morning and low at night. In older children, and in the slighter attacks of young children, there is but little fever, 100° F. being a common temperature.

When the larger tubes only are affected there is no *respiratory distress*, unless, in young children, the secretion be drawn into the finer branches. In severe attacks where the smaller tubes are involved the face is congested and cyanosed, the breathing much labored, and there may be considerable dyspnea. In such a case the respirations are rapid and gasping, the *alae nasi* dilate actively, and the pulse-respiration ratio is disturbed, an alteration to 3 : 1 being common, but not often greatly exceeded. There may be slight orthopnea.

The *skin* is hot and often dry at the height of the fever, but there is usually free sweating in the early morning hours when the temperature is low, and often at other times also. This is most marked, as a rule, about the head and face. While fever is present the skin over the trunk often appears flushed when exposed, with a slight punctiform accentuation, due, probably, to the activity of the sweat glands.

Bronchitis is often preceded or accompanied by catarrh of other mucous membranes. Nasal discharge is often noticed, and pharyngitis is not uncommon. These are especially to be observed when adenoids are present or when influenza is prevalent. In some cases there is conjunctivitis, and a suspicion of measles may be entertained until the fourth day is passed and no rash appears. Aphthous stomatitis is observed in some cases.

In older children there may be constipation; in infants there is very often some diarrhea throughout the attack, and sometimes preceding it.

*Physical Examination.*—In a severe attack the chest takes the position of inspiration owing to hyperinflation of the air cells, the result of dyspnea; therewith is often seen some inspiratory recession in the inframammary region owing to insufficient air entry at the bases, and this may become very marked, especially in cases associated with rickets. On palpation the hand placed upon the chest may often detect râles in the lungs; the movements are observed to be equal on the two sides unless in cases where there is considerable collapse at one base. Over the front of the chest the *percussion note* may be somewhat high-pitched, and occasionally some dullness over the middle lobe due to temporary collapse is found. Behind the note may be normal, but in infants there is often slight impairment at the bases caused by areas of superficial collapse, and sometimes such areas may also be made out over the rest of the lungs by light percussion.

*Auscultation* shows râles of various sorts and sizes scattered over the lungs. Thus, there may be abundant fine, moist râles audible both with inspiration and expiration, with, perhaps, a few sibili here and there; or the râles may be audible only, or mostly, at the end of inspiration; or at one point the râles are bubbling in character; or there may be only wheezy, dry sounds accompanied by croaking and purring sounds during expiration. As a rule, the râles are most abundant at the bases behind where they are mostly moist, the dry sounds being generally heard at the roots and upper parts of the lungs; the moist sounds are mainly formed in the smaller, and the dry sounds in the larger tubes, and both are generally present in the same case. The stage at which a case is examined does not determine the nature of râle that will be audible; moist râles are often heard a day or two after the onset and remain to the end, when they and the dry sounds in the larger tubes all clear up together.

The breath sounds are vesicular all over, though in front, over the emphysematous parts, they may be harsher than usual, and expiration somewhat prolonged. Behind, the breath sounds may be feebler at the base over the collapsed portions, but often the collapse is so superficial as to cause no diminution of the breath sounds, though the percussion note is muffled. In exceptional cases an area of collapse may extend sufficiently deep to give rise to bronchial breathing at some spot near the base, and in such a case the general symptoms and course must be taken into consideration to exclude inflammatory consolidation. The vocal resonance is not altered over the lung in bronchitis.

CAPILLARY BRONCHITIS is an inflammation of the finest tubes throughout the lungs, the process passing on in most cases into the alveoli, so that there is present at the same time an actual or potential bronchopneumonia. In the most acute cases death ensues before this disease has had time to become manifest. The symptoms are those of a bronchitis of exceptional severity; the child sits up in bed with the most urgent dyspnea, and in bad cases cannot afford breath either for feeding or crying. The surface is cold, cyanosed, and covered with sweat, the thorax appears prominent above from emphysema, with collapse at

corresponding recession at the bases. Râles are audible over the lungs, and the air entry is very deficient below. The condition is very fatal, the child becoming drowsy from carbonic acid poisoning, and passing rapidly to coma and death.

**Diagnosis.**—This is discussed under the heading of Bronchopneumonia (p. 626), the disease with which bronchitis is most liable to be confounded, and with which it may be associated.

**Prognosis.**—Acute bronchitis of the larger and medium tubes is very rarely fatal, apart from the advent of pulmonary collapse or bronchopneumonia in infancy. The disease, as a rule, proceeds to complete recovery, leaving behind, at most, some temporary emphysema and a susceptibility to bronchial catarrh. Capillary bronchitis, on the other hand, is very fatal, death being due to asphyxia often before the lesions of bronchopneumonia have had time to develop.

**Treatment. Immediate Treatment.**—The child should be put to bed in a room well warmed by an open fire, and efficiently ventilated either by a window opened at the top or by some other means. A stuffy, ill-ventilated room probably increases the risk of bronchopneumonia; the bed should stand away from the corner of the room; the temperature should be between 60° and 65° F., and the air kept moistened by a bronchitis kettle or by wet towels kept hanging before the fire. At the onset, in a severe attack, a calomel purge should be given, 0.06 gm. (1 gr.) to a child of two years, or 0.03 gm. ( $\frac{1}{2}$  gr.) combined with 0.06 to 0.13 gm. (1 or 2 gr.) of compound scammony powder to a child below this age. These can be dispensed with in a slight attack. In the early stages, when the cough is hard and dry, the simple expectorants, ipecacuanha or antimony, are of value to thin the secretion, and the following mixture may be prescribed for a child of one year, to be administered every three or four hours:

R—Vinî ipecacuanhæ . . . . .	0.3 c.c.	(℥v).
(or) Vinî antimonialis . . . . .	0.2 "	(℥ij).
Spiritus ætheris nitrosi . . . . .	0.3 "	(℥v).
Liquoris ammonii acetatis . . . . .	1.0 "	(℥xv).
Aq. chloroformi . . . . .	q. s. ad 4.0 "	(3j).—M.

If the child is very restless and ill, and especially if there is diarrhea, a few minims of brandy may be added with advantage, but the sedative effect of brandy must not be overlooked where cough is needed to clear the tubes of copious secretion.

When the secretion in the tubes is free, as indicated by the changed character of the cough, and not until then, the stimulating expectorants, ammonia, squill, and senega, may be given to help expulsion and control secretion; 0.03 gm. ( $\frac{1}{2}$  gr.) doses of ammonium carbonate may be given to a child of one year, as in the following prescription:

R—Ammonii carbonatis . . . . .	0.03 gm.	(gr. $\frac{1}{2}$ ).
Spiritus chloroformi . . . . .	0.07 c.c.	(℥j).
Infusî senegæ . . . . .	2.00 "	(3j).
Aq. . . . .	q. s. ad 4.00 "	(3j).

The object of treatment is to liquefy the secretion, and then to check its formation.

In cases where the tubes are blocked with secretion which cannot be expectorated, leading to deficient aeration of the blood, an emetic must be given. The most convenient is ipecacuanha, either the powder in 0.7 gm. (10 gr.) doses, or the wine, 4 c.c. (1 dr.) every one-fourth hour until vomiting is induced; this may be aided, if necessary, by tickling the fauces with a feather, or by the passage of the stomach tube.

*Prophylactic Treatment.*—If the health has been impaired by the acute attack, change of air, if the time of year allow it, to a dry place away from smoke and dust is a good beginning; but if it is a cold season of the year, as so often happens, this is not always advisable. The child must first be gradually acclimatized to cooler rooms and the ordinary air of the house, care being taken that he is lightly but warmly clothed with flannel next the skin, the legs completely covered, and the feet kept warm.

The child's bath should be given on rising in the morning instead of at night; at the latter time the skin may be rubbed briskly with a dry towel on going to bed. He should stand in hot water and be rapidly soaped all over, at the end a sponge full of cool water should be emptied down chest and back, and he should be taken out into a rough towel, and dried smartly until the skin is red and warm. After a time the cool water can be rendered cooler and cooler until it is used at the room temperature, but for infants under eighteen months of age 70° F. or thereabouts is generally cool enough, and lower temperatures may be depressing. If no reaction follows the cold douche it is evident that the child cannot stand it at the temperature given, and it must be suspended for a time. It is always better to begin this treatment in the summer weather; the cold douche can then generally be maintained throughout the winter months with advantage. The whole bath should not take above one and a half to two minutes from the moment the child steps in to the time he is taken out to be rubbed dry.

The living-rooms must not be kept overheated, and efficient ventilation must be secured. Except in very cold weather the window should be kept open at the top, and in winter the "poor man's ventilator," window board, is a very efficient mechanism, the lower sash being raised on a piece of wood which fits the window below, and allows a stream of air to enter between the sashes above. In French windows a ventilating pane is most convenient. The child must be out-of-doors as much as possible, a brisk run being permissible in nearly all kinds of weather if the best time of the day be chosen. If confinement to the house seems imperative the child should leave his nursery at least twice in a day, when the windows must be thrown wide open for efficient airing. He must be given as much sunshine as the season of year permits.

Wet shoes and stockings or damp clothing must be changed at once and dry substituted, the feet being rubbed with a rough towel if cold; cold feet are a common cause of catarrh of mucous membranes, and must be avoided. The foolish fashion of bare feet and sandals is responsible for many inflammatory attacks of mucous membranes, and is to be strongly deprecated in a cold and changeable climate. Cod-liver



oil will be of value in increasing the nutrition of the children, but no more good is to be expected from it than from plenty of nutritious food.

The desiderata, when all is said and done, are warm but light clothing and plenty of fresh air and exercise.

### CHRONIC BRONCHITIS.

In cases where catarrh of the bronchial tubes is neglected, and also where an unnatural susceptibility exists or is fostered by injudicious management, the condition termed Chronic Bronchitis is set up. The mucous membrane and its underlying structures become permanently damaged and thickened, and the secretion is excessive at all times, giving rise to the cough which leads to its expectoration. Often a less marked condition than this exists, the changes being less permanent, so that the cough disappears in the warm summer weather, to return again on the slightest provocation, the child being subject to exacerbations at various intervals. Sometimes the cough occurs only during the winter months, often in acute phases, lasting three or four weeks, with intervals of six weeks or two months between. Chronic emphysema is generally set up and may be much or little; if the former it adds both to the present distress and to the future susceptibility, and in such case the breath is short on exertion, the face congested, and the chest barrel-shaped. As a rule, the emphysema is moderate in amount.

**Symptomatology.**—During the acute attacks the conditions may be similar to those already described under Acute Bronchitis, save that to them are added the chronic disabilities of the affected organs. The inflation of the lungs is seen to be excessive, the heart unusually embarrassed, and, in rare cases, a tendency to finger-clubbing is noted. As a rule, the acute attack is rather a subacute exacerbation than a definite acute bronchitis, and the temperature is then but little or not at all raised. In cases where emphysema is marked some bronchial spasm not uncommonly accompanies the inflammatory change.

**Treatment.**—The treatment in the acute exacerbation is similar to that of acute bronchitis. When this is past, the danger of recurrence must be warded off. The child should winter in a warm climate, some dry, sunny spot being chosen, when possible, where he can be out of doors a great part of the day without danger of chilling. In such favorable surroundings there is hope, in childhood, that the mucous membranes will gain in resisting powers and the susceptibility be outgrown. If such change cannot be had the best must be made of the climate where the child lives, the child getting out-of-doors whenever the weather permits. A judicious and carefully graduated system of increasing the child's resistance must be carried out on the lines suggested under the heading of Prophylaxis in Acute Bronchitis. In addition, nourishing food, especially butter, cream, and eggs, healthy exercise, with precautions to avoid chills, and, at intervals, a course of tonics such as cod-liver oil with iron and creosote, or the hypophosphites of lime and soda, are necessary to

aid nutrition and enable the patient to outgrow his susceptibility. If the child has adenoids or enlarged tonsils they should be removed as a preliminary to all other treatment.

### PLASTIC BRONCHITIS.

This is a rare disease which is also described under the name of Fibrinous Bronchitis. It may occur at any period of life, many of the cases commencing in childhood.

**Pathology.**—This is not understood, but it forms a clinical entity apart from those cases where false membranes have been found as a result of irritating vapors, or associated with such diseases as diphtheria, phthisis, pneumonia, and certain of the infective fevers.

**The Casts.**—Large, round masses, yellowish green in color, are expectorated, which when placed in water separate into mucopus and the fibrinous cast of the tubes. This consists of a hollow stem generally about the diameter of a goose-quill, branching out into an arborescent arrangement representing the bronchi down to their finest ramifications. On other occasions fragments only of such casts are expectorated. Their color is grayish white, and they consist of a tough membrane whose main constituent is fibrin.

**Symptomatology.**—The attacks recur at intervals varying from a few days to many years, and the liability to them may continue for years, or throughout life. The onset may be with vomiting, and cough soon follows, accompanied in some cases by pain in the side. The cough is hard and dry, and, if the fibrinous exudation affects a large area of the bronchial tree, there may be considerable dyspnea, which continues until the membranes are expectorated. The respirations are rapid during the exudation period, the pulse-respiration ratio being disturbed to 3:1 or even  $2\frac{1}{2}$ :1 as in pneumonia. Many days pass before the casts separate, though some mucus may be expectorated, and after four days to one week or ten days, the large, round masses appear which contain the fibrinous plugs. Immediately thereafter the patient is easier, the dyspnea departs, and the temperature, which was high, drops to normal, to rise again with the formation of fresh casts, a process which continues for a variable period up to a fortnight or more. Relapse may occur with the involvement of fresh areas of the bronchial tree before the process quiets down.

In cases of extensive exudation the *physical signs* are marked, as in a child, aged six years, whom I have had the opportunity of observing during two attacks. In the first attack there appeared dulness at the left base up to the scapular angle, with feeble breath sounds and diminution of vocal resonance, but no added sounds—signs, indeed, of pulmonary collapse. Later a friction rub developed over this area, and, when the casts were loosened, some bronchial breathing with increase of vocal resonance, and sharp crepitations. In the second attack two months later the same signs appeared in the same situation,

and in a relapse which took place the right apex was attacked. A few râles generally appear after the membranes are loosened, and continue as long as any expectoration remains.

**Prognosis.**—The disease is not dangerous to life, though the liability to attacks may last for years.

**Treatment.**—The treatment is only expectant, since nothing is known to influence the disease. Biermer recommended the inhalation of a lime-water spray on account of the power of this drug, and of alkalies, generally, to dissolve the membrane. The patient should be kept in bed during the attack and the atmosphere be moistened by a steam kettle. Oxygen should be at hand in case the respiratory difficulty becomes urgent. Iodide of potassium and mercury have been recommended by some and emetics might possibly be useful in some cases.

### PULMONARY COLLAPSE.

**Etiology.**—Collapse of areas of the lung substance is of common occurrence in infancy, and adds a grave danger to all cases of bronchial catarrh. Its production depends largely upon the yielding nature of the ribs and cartilages in infancy, especially when rickets is present, as a consequence of which the muscular power necessary for inspiration is weakened, the inspiratory suction of the chest walls, whereby the lung is inflated, is feeble, and, in addition, the expiratory air-blast, by means of which the bronchial tubes are kept clear, possesses but little adequate expulsive power. The condition may arise with stenosis of the larynx, trachea, or bronchi, and not uncommonly occurs in quite mild cases of bronchitis, the secretion in the larger tubes being suddenly inhaled and causing blocking of a smaller branch, with subsequent collapse from absorption of the imprisoned air. Some amount of superficial collapse accompanies nearly all cases of bronchitis in infants, so that small, dark, depressed areas are found, postmortem, scattered through the lungs and alternating with areas of compensatory emphysema. Such areas give rise to neither physical signs nor noticeable symptoms. If more marked they cause some flattening of the percussion note down the back of the chest, especially at the bases, and their extent may be sufficiently definite to be mapped out by percussion, but if shallow they will give rise to no diminution of the breath sounds. Areas of collapse sufficiently large to cause symptoms commonly occur at the base and down the posterior border of the lung close to the spine, generally forming a wedge-shaped area with the base below. The middle lobe is a not infrequent seat of collapse, especially in the bronchitis of older children.

**Symptomatology.**—When considerable collapse occurs, the symptoms are of sudden onset and often develop during sleep. The infant has generally been noticed to cough and wheeze for a few hours or a day or two; perhaps he is already under treatment with a definite bronchitic attack. During the night he suddenly wakes screaming and fighting



for breath, the breathing is rapid and shallow, and the child becomes cyanosed, cold and collapsed, and very restless. Vomiting is not uncommon at the onset, and convulsions may occur. Death follows quite suddenly in some cases.

On examination marked inspiratory recession is noticed at the base of the chest along the diaphragmatic attachment, and may be more marked on one side than the other. Hyperinflation (acute emphysema) of the upper parts of the lungs occurs, partly as a compensatory effect and partly as a result of the violent inspiratory efforts, and, as a consequence, the upper part of the chest appears rounded and prominent. This superadded emphysema becomes as much an element in the threatening asphyxia as is the initial collapse. Over the bases of the chest, on one or both sides, where the affected area lies, the breath sounds are feeble or absent, and the expansion is diminished, but there is no dullness at first. It is only after the imprisoned air has been absorbed that the area becomes impaired or dull to percussion. Over the remainder of the chest bronchitic râles of various kinds will generally be audible.

**Diagnosis.**—When the collapse is considerable in extent the signs of pleural effusion may be simulated—dullness, absent breath sounds, and diminished vocal resonance. The history of onset in such cases, the less resistant dullness, and the signs of bronchitis over the rest of the chest will usually serve as distinctions. When the collapsed area is large in extent the neighboring viscera will move over toward it, and this forms a most valuable distinction from pleural effusion. Moreover, collapse is a disease of infancy, when effusions are generally purulent, and give signs like pneumonia rather than those likely to be confounded with collapse. It is but rarely that collapse is sufficiently massive to give the ordinary signs of consolidated lung (bronchial breathing and bronchophony). When such occurs it is generally at the apex, and the area is found at the autopsy to be engorged and edematous, and to present to the microscope the elements of a commencing pneumonia. Signs somewhat simulating inflammatory consolidation are occasionally found in bronchitis at the base also, and their nature can usually be determined by the slighter character of the constitutional symptoms and the rapidity with which the lesion clears.

**Treatment.**—This must be directed toward the removal of the obstruction in the tube, or, failing this, in stimulating inspiratory effort even at the expense of increasing the accompanying emphysema. Thus, an emetic may be given if the secretion in the tubes is abundant and loose. Ipecacuanha powder, 0.7 gm. (10 gr.), or wine of ipecac, 4 c.c. (1 dr.), every quarter of an hour, may be tried and the fauces tickled, or if these measures are unsuccessful the passage of a stomach tube will generally bring about the desired result. A hot mustard bath, 15 gm. to 4 litres ( $\frac{1}{2}$  ounce to 1 gallon), should be given, and the chest sponged with cold water to induce deep inspiration; the skin should be slapped until vigorous crying is induced, and liniments or dry-cupping applied over the bases of the lungs. Belladonna in large doses, 0.016 gm.



( $\frac{1}{4}$  gr.) of the extract, may be tried, both as a respiratory stimulant and for the purpose of drying up the bronchial secretion. The child must be roused at intervals and made to cry; it must be carried about the room and not allowed to sleep itself to death, as it will if permitted. Oxygen will be serviceable if given at intervals and, in some cases, artificial respiration may be needed.

### EMPHYSEMA.

This disease is dependent upon an overdistention of the lung alveoli. In some cases the lesion is permanent and associated with atrophy of alveolar walls, but in children the condition is often temporary, and, even where it has existed for many years, may disappear at puberty. For this reason the term Emphysema may be used to cover all cases, or may be reserved for permanent cases only, the latter cases being referred to merely as "hyperinflation." For clinical purposes, it is best to include all conditions of alveolar distention under the heading of Emphysema.

**Etiology.**—*Chronic emphysema* in children is always the result of bronchial catarrh. *Acute emphysema*, or hyperinflation, occurs in any condition of which dyspnea is a marked feature. It is especially common in acute bronchitis and bronchopneumonia, but also occurs with laryngeal obstruction, whooping-cough, asthma, and other respiratory conditions. It is a constant and serious accompaniment of pulmonary collapse, in which case it is, no doubt, in part compensatory.

No disease has given rise to more discussion with regard to its mode of origin than has emphysema. Two hypotheses are commonly advanced to explain its occurrence, the inspiratory and expiratory, to which is added "hereditary predisposition" by many. As a matter of fact two very different varieties of emphysema are observable, and there is good reason to believe that they are of different causation. One is the marked condition, often associated with the formation of air-containing bullæ, occurring in the unsupported portions of the lung, the anterior margins of the upper lobes, the edges at the bases, and often the extreme apex, and this is produced by the forces of *expiration*, as in violent and prolonged coughing. Interstitial emphysema is also of this type. The other variety is the general emphysema which leads to enlargement of the organ as a whole. This is probably due to violent *inspiratory* efforts the result of dyspnea, especially in such diseases as asthma and bronchitis, where expiration also is hindered by obstruction in the tubes. The condition itself depends on a loss of elasticity of the lung, whereby it tends to remain in the position of inspiration instead of returning to the normal position of rest. This loss of elasticity is due to overstretching, aided in many cases by inflammatory changes. There is reason to believe, also, that a congenital weakness of the elastic tissue of the lung leads to a more ready production of the disease.

*Compensatory emphysema* occurs whenever the volume of the lung is reduced, as by an area of fibrosis or collapse. Its production is purely

mechanical, and it probably compensates in no way for the loss of aerating surface caused by the condition which brings it about.

**Pathology.**—The morbid anatomy of this condition is similar to the condition found in the adult. The emphysematous lung is more voluminous than the normal. It is pale pink in color, soft to the touch, and the individual alveoli are plainly visible to the naked eye. In cases of longer standing there are, in addition to the simply over-distended air sacs, some larger blebs made by the fusion of a terminal air passage (infundibulum) with its surrounding alveoli, or of a number of air vesicles through rupture of their walls. (Wollstein.)

**Histology.**—In the mild, acute form, which is really only an overdistention, microscopic examination shows merely a dilatation of the air vesicles, and a consequent thinning of their walls with stretching or straightening of the capillary network. The walls between neighboring alveoli may be torn or entirely disappear.

In older more chronic cases atrophy of the elastic tissue and capillaries in the alveolar walls occurs, and neighboring alveoli, having lost their dividing walls through thinning and perforation, join to make large blebs. Such cases are rare in childhood.

**Symptoms.**—*Acute emphysema* most commonly occurs in acute bronchitis and bronchopneumonia; it is also present with pulmonary collapse and adds largely to the respiratory disability. The upper parts of the chest are very prominent, as in the position of inspiration, and there may be some recession at the base, along the diaphragmatic attachment. The percussion note is deep and full, the cardiac dulness covered, and the respiratory murmur harsh. The condition recovers completely when the cause is removed, though the lung may take some time to return to normal after a prolonged strain such as whooping-cough entails.

*Chronic emphysema* is rare in childhood; commonly the condition is slight, and the symptoms largely or entirely those of the accompanying bronchitis. The elements added by the disease itself are dyspnea, caused by the loss of respiratory surface, and cardiac disability, due to the straightened circulatory paths through the lung.

Dyspnea is sometimes considerable, persisting even through the summer months, when no bronchitis is present. In many cases it is only noticeable on exertion, except during the attacks of bronchitis, which so commonly occur, often in association with some bronchial spasm. The face is then congested and cyanosed, and the veins in the neck may be full or even pulsating. Some amount of finger-clubbing exists as a permanent feature in certain cases of long standing.

**Physical Signs.**—In a marked example, the chest is in the position of full inspiration and appears unduly rounded, the back being sometimes bowed. The angle of Ludwig is prominent, the clavicles project, the supraclavicular fossae being deep, or filled by the bulging apices of the lungs; the sternomastoids stand out, and the costal angle appears wide. The heart's apex beat may be difficult to palpate, and an epigastric pulsation is generally visible. The movements of the chest are confined—and consist mainly in an up-and-down, piston-like action.





and this is an association of bronchial spasm with attacks of bronchitis in childhood, this spasm being liable to recur with each subsequent bronchial catarrh. Such cases are familiar to most of us, the dyspnea generally lasting a day or two, and giving rise to considerable hyperinflation of the lungs. It is probably cases of this nature which for the most part swell the statistics of asthma in children, and, in accordance with this, one finds that all observers mention bronchial catarrh as an important causative factor. Thus 80 per cent. of Salter's cases originated with whooping-cough; 20 of Goodhart's cases had suffered with bronchitis, and many with measles and whooping-cough; 7 had had bronchopneumonia. Among La F  tra's cases 14 were assigned to bronchitis and 3 to varieties of pneumonia.

**Pathology.** The theory of spasm, so clearly proved by the recent work of Dixon and Brodie, is especially striking in view of the prevalence of adenoid growths in cases of asthma in children. It was found experimentally by these observers that the greatest reflex effect was obtained when the nasal mucous membrane, and especially the upper and posterior part of the nasal septum, was stimulated. La F  tra found adenoids present in 47 per cent. of his collected cases.

**Symptoms.**—Attacks may occur similar to those found in the adult, and in this case their symptoms merit no separate description. In cases of spasm associated with bronchitis it is not always easy to decide how far either of these elements is responsible for the distress which is present. The most noticeable feature is the marked dyspnea, generally accompanied by cyanosis, and the heaving respiratory movements of the barrel-shaped chest. The picture of the elderly bronchitic with emphysema is strikingly reproduced.

On examining the chest emphysema, or, more correctly, "hyperinflation," is very noticeable, the extraordinary muscles of respiration are in action, and the thorax moves up and down with a piston-like action. The lungs are full of wheezy sibili and rhonchi, which are peculiar in that they are audible alike with expiration as with inspiration. The duration of these attacks is measured not by hours, as in classical asthma, but by days.

**Treatment.**—The most important point in treatment is to remove the predisposition to those bronchitic attacks which so often initiate the bronchial spasm. For this the reader is referred back to the section on Bronchitis, where this matter is discussed. Change of climate may be necessary in some cases, and should certainly be urged where other means fail. At the same time any local disease, such as adenoids, must be scrupulously attended to.

For the attacks the nitrate and stramonium papers may be used as in adults, and where bronchitis is associated a steam kettle and wine of ipecac or wine of antimony are of great value. For the spasm nitroglycerin, 0.0006 to 0.0012 gm. ( $\frac{1}{1000}$  to  $\frac{1}{500}$  grain), every half hour for two or three doses, or atropine or belladonna, 0.017 gm. ( $\frac{1}{4}$  grain), extract, repeated till flushing of the face ensues, have often been successfully employed.



## CHAPTER XXV.

### BRONCHOPNEUMONIA—LOBAR PNEUMONIA.

#### BRONCHOPNEUMONIA.

**Etiology.**—The term Bronchopneumonia signifies a lung inflammation of certain well-recognized type, and as such embraces conditions of various causation. For purposes of classification, cases are conveniently divided under two headings, primary and secondary bronchopneumonia, the latter being again subdivided into many groups.

*Secondary bronchopneumonia*, as the name implies, comprises all such cases as are secondary to other diseases, the majority being either the outcome of an acute bronchitis of simple causation, or arising in the bronchial catarrh which so commonly accompanies certain of the specific fevers, more especially measles, whooping-cough and influenza; the remainder consisting of cases of septic origin, and those which terminate such diseases as marasmus, splenic anemia, and chronic diarrhea. Lobular pneumonia is, in most cases, a purely local disease of the lungs and lobular in distribution.

*Primary bronchopneumonia* comprises all such cases as arise without antecedent illness. It is generally caused by the pneumococcus, and is distinguished from the secondary disease by its abrupt onset and its occurrence in healthy children. In some cases the general symptoms are marked and a close resemblance to croupous pneumonia obtains, but, as a rule, the local symptoms overshadow those of general infection, and it is to the former that the anxious course and high mortality in bronchopneumonia are nearly always due. As a consequence of this, the symptoms of all varieties of the disease, whatever the cause, are sufficiently similar to be grouped together for purposes of description, being dependent so largely upon the local processes in the lungs.

The diagnosis of primary bronchopneumonia rests upon its sudden onset without antecedent illness, and it is open to conjecture whether two classes of cases are not included under this heading. It happens sometimes that an illness having all the features of a croupous pneumonia, with its sudden onset, high continuous fever, and short, definite course terminating by crisis in many cases, presents to examination all the physical signs of bronchopneumonia, or shows the lesions of that disease on the postmortem table. Such cases are undoubtedly general blood infections, and as such stand side by side with croupous pneumonia which they so closely resemble. They form but a small proportion, however, of those cases to which the term primary bronchopneumonia is applicable, and it seems probable that the remainder are

of different origin, being due probably to a sudden invasion through the small bronchial tubes, a capillary bronchitis, whether scattered in distribution or general. To put it differently, in one class the lung is invaded from the blood stream and in the other from the air tubes; the distribution of blood and air to the pulmonary lobule being similar the lesions produced are identical. This is well illustrated by pulmonary tuberculosis in which the lobular distribution of acute phthisis may be exactly imitated, in isolated areas, by the blood invasion of miliary tuberculosis.

I have attempted to tabulate a series of cases of bronchopneumonia, 236 in all, to show, at any rate roughly, the proportions in which the primary and secondary cases occur:

Primary . . . . .	80 cases or 34 per cent.	Mortality 60 per cent.
Secondary . . . . .	156 cases or 66 per cent.	
" to bronchitis . . . . .	58 cases.	" 64 "
" "whooping-cough" . . . . .	28 "	" 50 "
" "measles" . . . . .	38 "	" 55 "
" diphtheria . . . . .	7 "	" 100 "
" diarrhea . . . . .	13 "	" 100 "
" sepsis . . . . .	11 "	" 100 "
" marasmus and congenital syphilis . . . . .	8 "	" 100 "
	156 "	

**Incidence.**—Bronchopneumonia is a disease of infancy and is commonly met in children above the age of three years, the majority of the cases probably occurring between the sixth and twelfth month of life. It is most prevalent during the winter months, corresponding in this respect, as might be expected, with bronchitis. It occurs most widely among the children of the poor and is especially prevalent in overpopulated areas.

**Pathology. Bacteriology.**—Most of the workers on this subject have failed to separate the primary and secondary cases. The only complete account on these lines is that given by Dr. L. Emmett Holt in his book on diseases of children from cases collected by Dr. Martha Wollstein. From this it appears that 76 per cent. of the primary cases were caused by the pneumococcus, the other organisms found being mainly streptococci and staphylococci. Among the secondary cases, in 64 per cent. the pneumococcus was present, but the majority were due to mixed infection, a streptococcus being found in 37 per cent., the other organisms present being of various kinds and including such varieties as staphylococcus, bacillus diphtheriae, bacillus pyocyaneus, and bacillus coli communis. The streptococci were associated in especial frequency with cases secondary to the infectious fevers.

**Morbid Anatomy.**—The consolidation may be scattered, or may involve large areas, even the whole of a lobe, in which case the affected part will appear voluminous and the pleural surface roughened, showing petechial hemorrhages and covered with a thin layer of fibrin. The appearances of the bronchopneumonic parts are very various, the consolidated areas generally appearing as light points off a background.

<sup>1</sup> A higher age average was probably accountable for the lower mortality among these cases.

of dark-brown, congested lung. They are the size of pinheads or larger, and are arranged, some in a circle round a small pus-containing bronchus, some in clumps showing no relation to the air tubes, some themselves pierced by a minute bronchus and forming a peribronchial nodule. In massive consolidation the whole surface appears firm and smooth, red marbled with various shades of gray.

With regard to the color of the pneumonic patches this varies greatly according to the form of microscopic element composing it. No division into stages can be made as in croupous pneumonia. The areas are usually slightly raised and reddish-gray in color; where there is much leukocytic infiltration they appear whiter. In acute cases the areas are small and gray or yellowish-white in color and the tubes contain mucus; when the process is earlier still, bronchitis with minute dark points of collapse may be seen. In cases of long continuance the areas are usually large, and greenish or yellowish in color, and the small tubes contain pus. In many such chronic cases the larger bronchial tubes are dilated at the root and in the lower lobes, and occasionally the finer divisions also. Small abscesses may be found scattered through the lungs, especially just beneath the pleural surface. They are generally formed round the walls of a small bronchus through softening of a peribronchial nodule; in some cases the pus in a localized dilatation of a bronchiole appears like a minute abscess.

It is common to find much collapse in the lower lobes, especially, I think, in cases of diphtheritic origin, and also some emphysema of the upper and anterior parts of the lungs. The septic cases tend, in my experience, to be lobar in distribution, an upper lobe being not uncommonly attacked, and there is usually considerable engorgement and edema of the less affected portions of the lungs.

**Histology.**—Much variety is found in the microscopic appearances. The small bronchi contain plugs of epithelial cells, or of polymorphonuclear leukocytes. The most infiltrated areas generally surround these bronchi, but may be scattered elsewhere. These areas usually consist of dense masses of leukocytes, both filling the alveoli and also infiltrating the bronchial and alveolar walls. The interalveolar capillaries are engorged and patches of collapsed air cells are seen here and there. Other areas are found in which the alveoli are filled with blood cells and with granular fibrin and serum; their epithelium is swollen in places, and in parts of the section the alveoli may be seen filled with desquamated lining cells. In cases where the process has been of long duration the alveolar walls are thickened with proliferated connective-tissue cells, an earnest of that process which in some cases leads on to a widespread fibrosis.

**Symptomatology.** *Onset.*—As the symptoms of onset of the primary and secondary varieties of the disease are somewhat different it will be necessary to describe these separately.

*Primary Bronchopneumonia.*—Cases occur, as already indicated, which are indistinguishable from croupous pneumonia save for the difference of the lesion found in the lungs. The symptoms of these



cases need no further description here; it suffices to remember that all the symptoms of croupous pneumonia may occur with a bronchopneumonic lesion.

In the remainder the onset is generally abrupt; it may be less sudden and more accurately described as "rapid." The *respiratory difficulty*, *fever*, and *cough* are generally the first things noticed, though some more definite symptom such as vomiting may occur at the start. Convulsions are but rarely seen and a rigor almost never. The skin may be dry and burning, as in croupous pneumonia, but often there is sweating. The bowels may be costive at the commencement, but diarrhea nearly always follows, and may obtain from the beginning. As in croupous pneumonia robust children are attacked, which less often happens in secondary cases.

*Secondary Bronchopneumonia.*—In those cases which follow on bronchitis of the larger tubes, there occur first the ordinary symptoms of this disease, cough and fever, sometimes accompanied or preceded by nasal and pharyngeal catarrh, and these symptoms either gradually increase, or, more often, after they have lasted a variable time, the child suddenly becomes worse, the cough gets dry, hacking and distressing, the respiration rapid and difficult, and in a short time all the symptoms of bronchopneumonia become evident.

In cases following the specific fevers the bronchopneumonia may either begin during their course or more often there is an interval of a week or two during which the bronchitis continues, and then gradually increases in severity to pass into the more serious disease. Secondary bronchopneumonia from the conditions under which it occurs is usually found in sickly and ill-nourished infants.

*General Symptoms.*—After the onset is past and the disease fully established, the symptoms are closely similar in the primary and secondary cases; where differences occur these will be pointed out.

The child shows a flushed face in the early stages; later it may appear pale. The skin is hot and moist, sometimes dry and burning in primary cases; the respirations are rapid and the *alae nasi* work. In many cases there is slight cyanosis of the lips and ears, and when distress is more urgent there may be a leaden tint over the whole face. The child is restless and irritable and refuses food, but is thirsty; in infants the breast is often refused owing to the urgent needs of the respiratory system. Diarrhea generally occurs, and occasionally vomiting with the cough is troublesome. Nasal discharge is often observed and conjunctivitis in some cases. Stomatitis and pharyngitis are not common.

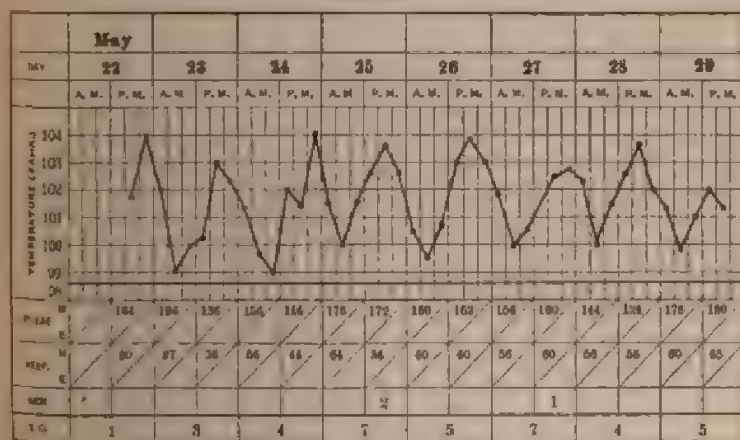
The *cough* in bronchopneumonia is generally a more important feature than in croupous pneumonia; it is dry and hacking and often very frequent and distressing. There is no expectoration in children, but sometimes some viscid mucus is brought into the mouth. Vomiting is not infrequently accompanies the cough, both at the commencement and also later when the cough is looser or sometimes spasmodic, as may be even in cases which do not accompany whooping-cough. The



sleep is often disturbed by cough, and the attacks cause a temporary hueness round the lips and eyes. In cases following whooping-cough the characteristic cough may disappear during the attack and only return with convalescence.

The *dyspnea* is often extreme and depends chiefly upon the amount of the accompanying bronchitis of the small tubes. The respirations are rapid, and the pulse-respiration ratio disturbed. In some cases this ratio is reduced to 2 : 1, more often only 3 : 1, but in slight cases it may be but little altered from the normal, and does not form a valuable point of distinction from cases of bronchitis. The breathing is often accompanied by a short grunt or gasp at the beginning of expiration, though this is usually a less noticeable feature than in croupous pneumonia. Where there is much respiratory difficulty the *ala nasi* work, and, if any cyanosis is present, they will be seen to dilate vigorously. In slight or chronic cases where breathing is not much disturbed they

FIG. 132



Temperature chart, case of bronchopneumonia.

may be quite inactive. When the amount of air reaching the lung alveoli is much reduced there is distressing dyspnea. The child is deeply cyanosed, with a cold, damp skin, and the whole attention is given to the respiratory functions; if the condition is not relieved the lung becomes more and more choked, tracheal râles appear, drowsiness supervenes, and as the cyanosis increases unconsciousness and death soon follow.

The temperature shows wide variations in bronchopneumonia and is perhaps more inclined to be high in primary than in secondary cases. But seldom is the temperature so high as in croupous pneumonia, 101° to 103° F. being a common evening standard, with a drop of 3° or 4° by morning (Fig. 132). This swinging temperature goes on throughout the attack, and is as much a feature of the primary as of the secondary cases. In slight cases the temperature sometimes only rises to 99° or 100° F. and falls to 97° or 98° F. in the morning; sometimes

fluctuations of only  $2^{\circ}$  are observed. In rare cases the temperature remains normal throughout; these are cases with marked wasting and are not of favorable outlook. Hyperpyrexia but rarely occurs in bronchopneumonia unless as a terminal event.

Generally the disease subsides with a gradual lowering of the temperature extending over many days or weeks, but occasionally a crisis occurs as in croupous pneumonia, and that in secondary as well as in primary cases. As a rule, the temperature makes a few swings after the critical fall, and very rarely presents the continuous subnormal temperature so commonly found for a few days after the crisis in croupous pneumonia. Moreover, the respirations do not drop to normal so rapidly thereafter in bronchopneumonia.

In the primary cases the *skin* may be hot and dry as in croupous pneumonia, and remain so throughout. In most cases there is sweating and this is always a feature of the secondary cases. It is often profuse when the temperature falls in the early morning, and generally most noticeable over the head and face. In the rickety children so often the subjects of bronchopneumonia it is very marked, but marked sweating occurs apart from rickets. In some cases of bronchopneumonia with considerable wasting, patches of fine, close-set petechial spots may appear over the abdomen, forming purplish patches which are of very fatal omen.

*Diarrhea* is an almost constant accompaniment of bronchopneumonia whether primary or secondary, and is often severe. It sometimes precedes it and is probably then a predisposing cause of the initial bronchitis. Four or five loose motions daily are commonly passed, and these often contain undigested food and are very offensive. The diarrhea often adds considerably to the gravity of the case. *Vomiting* is an occasional initial symptom; more commonly it occurs as a result of coughing and thereby assists in the expectoration of phlegm.

*Nervous symptoms* are much less commonly seen than in croupous pneumonia and are of more serious import. Convulsions occasionally occur, or twitchings of the facial muscles and passing strabismus, generally at the onset in primary cases where the general poisoning has been marked. Now and then a case is met where the symptoms from the beginning suggested a vertical meningitis, and where at the autopsy nothing but bronchopneumonia appears, as in a child who was convulsed for sixty hours up to death and in whom bronchopneumonia was the sole lesion discovered.

**Physical Signs.**—These vary greatly in different cases. They are at first those of bronchitis only and may remain so throughout the attack but generally some definite signs of consolidation appear. The signs are nearly always most marked in the lower lobes behind, and very rarely does consolidation occur at an apex except where other parts of the same or opposite lung are also involved.

On *inspection* it is generally noticeable that the upper parts of the chest are prominent and the thorax held in the position of inspiration; this is due to a hyperinflation of the lungs, a temporary emphysema.

caused by the inspiratory dyspnea. It is accompanied in many cases by some inspiratory recession at the base of the chest, and this may be very marked when the obstruction in the small tubes is great. Rickets is very commonly present in cases of bronchopneumonia, and a deformed chest with submammary groove, wide epigastric angle, and a thrusting forward of the lower rib cartilages is often to be observed.

*Palpation and Percussion.*—Where the acute emphysema is marked the heart is largely covered and the apex beat may be difficult to find, but it can generally be palpated either in the normal or just outside the normal situation. The percussion note over the front of the chest may appear somewhat boxy and hyperresonant; behind it is often poor

FIG. 133



Child with bronchopneumonia.

over both sides from the presence of small areas of collapse, especially at the bases. Such signs are those commonly present in bronchitis, but, in addition, there generally appears more definite dulness at the bases behind, due to the presence of areas of lung consolidation. The dulness may be slight with a high-pitched note; occasionally it is marked with increased resistance to percussion, and involves the greater part of a lobe so as to simulate the consolidation of croupous pneumonia (Figs. 133 and 134).

*Auscultation.*—Over the front of the chest the breath sounds may appear somewhat harsh on account of the emphysema; commonly they are not appreciably altered from the normal.

Râles of various sorts and sizes may be audible scattered over the



lungs, often moist in the small and dry in the large tubes. Often the râles are confined to the bases behind. These are the signs of bronchitis and in the early stages nothing more definite may be found, but later signs pointing to consolidation also appear. First, there may be heard at the bases behind fine or medium-sized râles with the peculiar sharp, resonant, metallic quality which denotes the presence of lung consolidation around the tubes. Secondly, there may be signs pointing to definite areas of consolidation, mostly at the bases behind. In such case bronchial or tubular breathing will be heard, generally intense and

FIG. 134



Bronchopneumonia. Girl of sixteen months; dots represent crepitations and the lines represent percussion dullness.

close to the ear, and accompanied by showers of the sharp metallic râles above described. The vocal resonance is increased over these areas, often to the extent of bronchophony. When bilateral, these signs are generally more marked at one base, and often there is definite consolidation found at one base and only metallic râles at the other. Sometimes one base only is affected when the signs of croupous pneumonia are simulated, but in such cases the râles in the consolidated area are generally more abundant than are found in that disease. In some cases definite signs of consolidation never appear, and a diagnosis from bronchitis has to be made on other grounds to be mentioned here.



after. In addition to the râles, a fine, superficial pleural rub may be audible over the consolidated areas.

**Course.**—The attack lasts a variable time and is not self-limited as in croupous pneumonia, except, perhaps, in certain rare cases of primary bronchopneumonia. Between two or three weeks is an average duration in a favorable case, and the signs in the lungs usually remain another ten days or so. The dulness becomes less marked and gradually clears, the bronchial breathing changes through bronchovesicular to harsh breathing in which expiration is rather loud and prolonged, and from this to normal. The last to disappear are the râles, moist or dry, which remain for some days after the consolidation has entirely cleared, accompanied in some cases by weakness of the breath sounds.

**Clinical Varieties.** *Protracted Bronchopneumonia.*—Cases of very long duration occur often after measles or whooping-cough and generally end in death, but may clear up either partially or completely. Some of these cases begin acutely and run a high temperature for the first few weeks; some are indolent from the outset and show a moderate temperature range and rather subacute symptoms. The child wastes steadily, the signs persist or spread slowly, and a suspicion of tuberculosis is raised or even a diagnosis of that disease made. In some cases the signs nearly clear, only some impairment being left perhaps at the bases, but the child does not improve and continues to waste. Diarrhea is common, the finger-tips become glazed and perhaps clubbed, the eyelashes grow long; the skin gets harsh, dry, and yellowish, and often becomes covered with a growth of downy hair, especially down the back. Groups of fine petechial spots in the skin of the abdomen sometimes appear, as already mentioned, in such cases.

All these signs accompany a tuberculous bronchopneumonia with great certainty, but the same are found in a protracted bronchopneumonia of other causation, and errors of diagnosis between the two diseases are extremely common. In many cases, indeed, a differential diagnosis is quite impossible without a careful examination of the mucus from the back of the throat for tubercle bacilli. Two or three months is a common duration for a protracted bronchopneumonia, and at the autopsy a dilated bronchial tree with some fibrosis around it and at the root of the lung are found, besides more or less bronchopneumonic consolidation of old or recent standing. In cases which recover the return to health is slow and, as regards the lung, often incomplete.

*Bronchopneumonia Secondary to the Infective Fevers. Measles.*—These cases are often of long duration, but may be acute like those of other causation. In many of them the consolidation is lobar in distribution, and in fatal cases wide areas of moist gray or pink consolidation are found at the autopsy.

*Whooping-cough.*—In these cases the whoop often disappears during the attack to reappear with convalescence, though the cough may remain spasmodic and exhausting. The child may show the puffiness

of the face so common in whooping-cough and, if the bronchopneumonia comes late, the exhaustion from the original disease will make the prognosis more serious. Bronchiectasis and fibrosis of the lung are common sequelæ of these cases.

*Diphtheria.*—Nearly all cases secondary to diphtheria are fatal. They commonly follow laryngeal diphtheria, and are, hence, found after intubation and tracheotomy. They are generally caused by a spread of the diphtheritic process down the bronchial tubes and the diphtheria bacillus can be isolated from the lung in a proportion of these cases, but some are septic and due either to an unhealthy tracheotomy wound or to inhalation of foreign particles during feeding.

*Septic cases* are of necessity fatal; their causes are various. Among the cases tabulated under "etiology" the following primary lesions were found: Retropharyngeal abscess in two, abscesses elsewhere in two, a suppurating umbilicus, a suppurating patent urachus, ulceration due to a foreign body in the esophagus, and a suppurative nephritis.

Cases following *athrepsia* and *congenital syphilis* or *chronic diarrhea* are of bad prognosis, but are generally overlooked; the symptoms are often suppressed and atypical, the disease being only a terminal infection. In such cases the pulse-respiration ratio should draw attention to the condition. Some of the diarrheal cases show ulcerative colitis at the autopsy.

*Complications.*—The complications of bronchopneumonia are mainly infective lesions of various kinds and are generally of fatal termination. *Otitis media* is the least serious among them, and the remainder, *empyema*, *purulent pericarditis*, *purulent peritonitis*, and *purulent meningitis*, are still more common complications of croupous pneumonia and will be discussed under the heading of that disease. *Abscess* and *gangrene* of the lung are described under separate headings. In addition, I have found *cellulitis* of the chest wall as a complication. Bronchopneumonia when it leads to death is generally fatal on its own account, complications being found in but a small proportion of cases.

*Sequelæ.*—The commonest and most important sequelæ of this disease are bronchiectasis and pulmonary fibrosis. In some cases the bronchiectasis disappears, but in others a permanent dilatation of the tubes remains.

*Diagnosis.*—When a case of acute disease presents itself with cough and shortness of breath, accompanied by fever and malaise, our attention is naturally drawn to the lungs as the source of the symptoms, and we expect to find one of three conditions—either bronchitis, bronchopneumonia, or croupous pneumonia. It is, then, from the first and last of these that bronchopneumonia must be separated. In addition, the bronchopneumonia may be simple, or of tuberculous origin.

*Bronchitis.*—From acute bronchitis it may be diagnosed both by symptoms and by signs, though in certain cases the distinction is difficult or even impossible.

The onset may be similar in the two diseases and helps but little; the pulse-respiration ratio may be of some assistance, the disturbance

of respiration being generally greater in bronchopneumonia, and if the ratio is altered as far as 2 : 1 this disease is probably present; a ratio of 3 : 1, on the other hand, is found in bronchitis, but only with extensive bronchitic signs. The nature of the cough affords little help, and dyspnea and cyanosis are merely an index of the bronchitis which occurs in both. The temperature is sometimes of importance on account of its different duration in the two diseases, but is of no value at the outset. It may be equally high in both, but in bronchitis it generally lasts a few days to a week, whereas, in bronchopneumonia the course is considerably longer. Diarrhea may occur in either disease; constipation is seldom found with bronchopneumonia, but sometimes occurs with bronchitis, though generally in older children. The age of the patient is of assistance since bronchopneumonia is a disease of infancy, cases above the age of three years only occurring occasionally as a secondary affection. Nervous phenomena are rarely seen in bronchitis, and, as a rule, the patient does not appear so acutely ill as in a case of bronchopneumonia; this is often an important point.

The early signs in bronchopneumonia are those of bronchitis only, and occasionally in cases diagnosed on other grounds no signs pointing to consolidation appear in the lungs. The bases behind are the position at which such signs should be sought. They may be indicated by sharp, resonant râles of a metallic quality, no further signs of consolidation being obtained; but generally at some spot dulness, bronchial breathing, and bronchophony appear in addition to the fine metallic râles mentioned above. These nearly always indicate inflammatory consolidation, but occasionally the breath sounds may be bronchial or approaching bronchial in quality over an area of firm collapse in bronchitis only. In this event a diagnosis of bronchitis will generally be possible on other grounds; the constitutional symptoms will be less acute, and the suspected area will clear up with greater rapidity.

*Loobar Pneumonia.*—The differential diagnosis will be discussed under the heading of that disease (p. 641.)

*Pulmonary Tuberculosis.*—Two forms of tuberculosis may simulate bronchopneumonias of different types. Firstly, *acute miliary tuberculosis* may be difficult to distinguish from cases of acute bronchopneumonia, the lung symptoms being often prominent before those of the meningitis so commonly present arise. In such a case the child is acutely ill, and the lungs are filled with fine, moist râles, together with areas of consolidation in some cases. Usually the case presents a more hopeless aspect than does one of simple bronchopneumonia. The child is often poorly nourished, the skin is cyanosed, with a dark flush sharply confined to the cheeks, the spleen and liver are often large and firm, and, as a rule, meningeal symptoms soon appear—drowsiness, twitchings, sudden screamings, and, perhaps, some head retraction.

Secondly, protracted bronchopneumonia of simple causation is sometimes indistinguishable from *chronic pulmonary tuberculosis* in young children, this being indeed a bronchopneumonia of specific causation.



In such cases the disease spreads by the lymphatics from caseous nodes at the root of the lung, and consequently the most marked signs, and those first to appear, are in the intrascapular region behind, more often on the right side. This distribution, the tendency of the signs to persist and spread, the physical signs of softening of lung deposits, when such occurs, and the signs of tubercle elsewhere, as in peritoneum, intestine, spleen, etc., may give the clue to the nature of the condition. Such general symptoms as wasting, dry skin, petechial hemorrhages, growth of eyelashes and body hair, finger clubbing, or even some edema of extremities may occur in both the simple and the tuberculous disease; edema more often accompanies the latter.

**Prognosis.**—This is very serious in all cases of bronchopneumonia. Some idea of the mortality among hospital cases may be got from the table appearing under etiology. The disease is much less common among the well-to-do classes, and is much less fatal. It will be seen that the mortality appears higher among the primary cases and those due to bronchitis than among those secondary to measles and whooping-cough. This is, I think, due to a somewhat higher age average among the latter cases. Age is a very important factor in prognosis, and next to it comes the general health and nutrition of the child, especially with regard to the presence of rickets. Diarrhea is a serious complication when severe, and should receive prompt treatment. It will be observed that cases secondary to athrepsia, congenital syphilis, and diarrhea are nearly always fatal, being often merely a terminal infection. Those accompanying diphtheria and septic lesions owe their hopeless aspect to the nature of the original disease.

**Treatment.** We have no specific treatment for this disease. That which comes nearest to it is the treatment with full doses of belladonna, after the manner recommended by Dr. J. A. Coutts. The drug is given in doses of 0.016 gm. ( $\frac{1}{4}$  gr.) of the extract to infants and young children, repeated every two or three hours. These doses cause marked flushing of the face, thirst, and often some pœvishness, but symptoms of poisoning are rare and never serious. The drug is especially valuable in cases where bronchitis of the small tubes is a marked feature, and acts, probably, by controlling the secretion and thus clearing the small tubes, and also as a stimulant to the respiratory centre. The results are very striking in many cases, and it should always be given a few days' trial. Small doses are useless.

In cases of capillary bronchitis, or where the secretion in the tubes is abundant and is adding to the dyspnea, an emetic should be ordered at the start. Ipecacuanha wine in 4 c.c. (1 dr.) doses, or ipecacuanha powder in 0.6 gm. (10 gr.) doses repeated in a quarter of an hour, if necessary, may be given, assisted, if necessary, by tickling the fauces with a feather, or by passing a soft stomach tube. Vomiting is sometimes impossible to induce in these cases.

Counterirritation of the chest is useful in cases where pulmonary collapse is suspected, and where there is much bronchitis. It may be applied either by stimulating liniments, or with a mustard or Chloro-



paste. This may be reapplied from time to time as indicated. It is questionable whether cotton and wool jackets do more than add to the difficulties of breathing.

Fever must be controlled if excessive, but antipyretic treatment is less often required than in croupous pneumonia. Water in some form is the best antipyretic, and its application will be discussed when croupous pneumonia is considered. It must be used with caution for infants, as their vitality is easily depressed, and its application must be considered dangerous and useless in cases where the skin is not well flushed with blood. It is essential that there should be a reaction after bathing. When the surface is cold and the rectal temperature high, a bath at 105° to 110° F., with or without mustard, 15 gm. to 4 litres ( $\frac{1}{2}$  oz. to 1 gallon), will bring the blood to the skin and reduce the fever. A dose of brandy in hot water will assist this action.

Many cases of bronchopneumonia, including all secondary cases, are in need of stimulants throughout, and of these whiskey and brandy are the most satisfactory. They may be given in quantities up to 15 to 30 c.c. ( $\frac{1}{2}$  or 1 oz.) daily to a child of one year, and this is best divided into small quantities every three hours. Strychnine and caffeine are indicated where heart failure seems likely to occur, and digitalis is recommended by many.

Diarrhea must be treated by appropriate feeding. Careful feeding will in many cases be the most successful method of management. The milk must be well diluted, as with barley-water, and may be previously digested with pepsin or pancreatin, or 0.065 gm. (1 gr.) of papain with 0.15 to 0.2 gm. (2 or 3 gr.) of bicarbonate of soda may be added to each feeding. In some cases albumen-water (the whites of two or three eggs to one pint of water) may be substituted for milk during twenty-four hours or more. A dose of castor oil, followed by a mixture containing 0.15 to 0.2 c.c. (2 or 3  $\mu$ ) of the same, with 0.03 c.c. ( $\frac{1}{2}$   $\mu$ ) of tincture of opium (for a child of one year) to each dose may be effective.

At the termination of the attack, or in persistent cases where the temperature is low, tonics such as quinine combined with small doses of iron should be given. Some of the protracted cases are much benefited by being carried into the open air when weather and season permit. In any case the child should be moved to the seaside as soon as he is well enough to travel.

#### LOBAR PNEUMONIA

Lobar or Croupous Pneumonia, the common pneumonia of adult life, is still more common in infancy. All recognize its occurrence after infancy is past, but some, up to the present, fail to realize its prevalence during the first two years of life. The reason for this is twofold. First, that the disease is but seldom found in the postmortem room. This is explained by the low mortality of lobar pneumonia in childhood, and also by the fact that when death does occur it is generally due to com-

plications, the original lesion having had time to clear up. Second, because cases in which a diagnosis of lobar pneumonia was made not infrequently show after death the lesions of a bronchopneumonia with lobar consolidation. This is due in part to the difficulty, amounting to impossibility in certain cases, of diagnosing between these two conditions, but also in part to the fact that bronchopneumonia is not infrequently a fatal complication of lobar pneumonia in infancy. When this occurs, death is generally delayed until long after the lesions of the original disease have entirely disappeared.

The accompanying chart shows the relative frequency of the disease at different years of life among 196 cases collected by me. It will be observed that the greatest number occur at the age of two years, the

FIG. 135

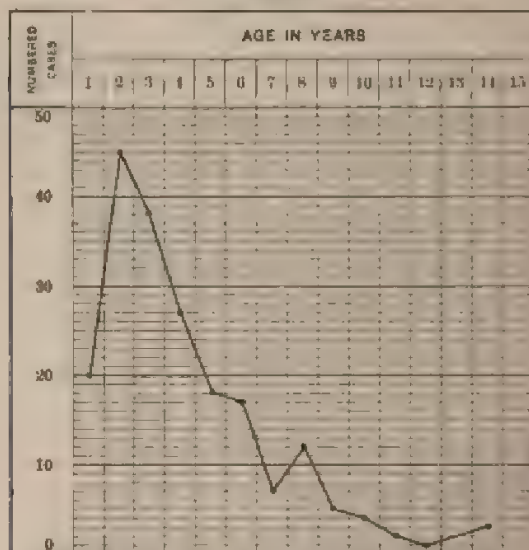


Chart showing relative frequency of lobar pneumonia.

disease becoming less and less frequent with advancing years. Pneumonia, including all forms, is of very common occurrence in infancy and 25 per cent. of the cases, at a low estimate, are instances of lobar pneumonia (Fig. 135).

**Etiology.**—Lobar pneumonia is a primary infective disease and never secondary; the micro-organism present is in most cases pneumococcus, and some predisposing cause can generally be discovered in association with its occurrence. A definite history of exposure is occasionally obtained, and cold is probably instrumental in lowering resistance. Trauma is an occasional factor. One attack of pneumonia predisposes to subsequent ones in children as in adults, and in old children it is not uncommon to obtain a history of one or more seizures in the past. It is sometimes, but rarely, epidemic.

**Morbid Anatomy.**—The appearance of the lung in lobar pneumonia of infancy and childhood is that of the same disease in the adult, save that when hepatization is established the cut surface is less coarsely granular, owing to the smaller size of the alveoli, and the outline of the lobules is more distinct.

**Histology.**—The microscopic appearance is similar to that found in the adult, but the exudation is granular, consisting of fibrin and serum.

**Symptomatology.** *Onset.*—The onset of the disease is sudden, or at least rapid in nearly all cases. The most common initial symptom is *vomiting*; this generally occurs once or twice only, but occasionally it continues for the first few days after food. *Chilliness* is not uncommonly complained of, but a *rigor* is distinctly rare in childhood, in contradistinction to its prevalence in adult life. *Convulsions* are uncommon at the onset of pneumonia, even in young infants, but they may occur, as in the case of a child of one and a half years of age in whom four convulsive attacks occurred during the first day of the disease. *Rapid breathing* is one of the earliest changes noticed, and often directs the attention of the friends to the chest as the cause of illness. In many cases the *alæ nasi* are noticed to be overactive. In children old enough to indicate its presence *headache* is usually present, sometimes lasting for days, and *pain* in the side is often complained of. This is generally thoracic, but is often abdominal, and may simulate that of appendicitis, peritonitis, or some other abdominal disease, as in the case of a young child where the pain was paroxysmal and led to a suspicion of intussusception.

*Cough* is generally slight, and is often overlooked by the parents and friends; it is seldom distressing or frequent as in bronchopneumonia, but it may aggravate the pleural pain and cause the child to scream or cry out at intervals. It is dry and hacking in quality, short, and purposeless, and is unaccompanied by expectoration. Quite commonly the cough does not appear until several days after the onset, and in a few cases it may be absent throughout.

Accompanying these definite symptoms are others more general. The child is drowsy and languid, and shows a disposition to be nursed, and in young children the power of walking is often lost temporarily. Food is refused, but thirst is intense, and cold water eagerly demanded. The child avoids the light and is peevish if roused, sleep is disturbed, and he is restless at night. A general tenderness of the body to handling is found in some cases.

*Constipation* is an interesting feature of lobar pneumonia; it is found in all older children and also very generally in infants, in contradistinction to the prevalence of diarrhea as an accompaniment of bronchopneumonia. It may even occur, as I have had opportunity to observe, in a child whose motions were normally loose.

A robust child, then, to take a typical example, is suddenly seized with the symptoms already mentioned. He presents to observation a flushed skin, with heavy red color on the cheeks and bright, shining eyes, often with some anxiety of expression. The tongue is thickly

furred and there may appear some herpes at the side of the mouth. Certain symptoms must be reviewed in more detail.

*Dyspnea.*—The breathing is very rapid, and the alae nasi work vigorously. The respirations are often 50 or 60 per minute, and may reach 80 or 100 in a young child. The normal pulse-respiration ratio is disturbed generally to 3 : 1, but occasionally to 2 : 1 or even  $1\frac{1}{2}$  : 1. This disturbance is of value before the lung signs have appeared, but later is not such a valuable diagnostic feature in childhood as in adult life, since it occurs also, to a great extent, in other severe respiratory affections. In slight attacks of lobar pneumonia the pulse-respiration ratio may be but little disturbed. The quality of the respirations is as peculiar as is their rapidity. There is a distinct pause at the end of inspiration, and expiration is accompanied by a grunt, or occasionally a groan. In some cases this grunt becomes a short cough, and is then very distressing and leads to much exhaustion. Though the respirations are rapid there is often no distress, but if, as rarely happens, the available respiratory surface is greatly reduced there is cyanosis, and the child lies propped up in bed with an anxious eye, all his attention being expended on his respiratory functions. Such conditions of marked dyspnea are more often observed in bronchopneumonia.

*Skin.*—The skin is hot, dry, and pungent to the touch, a condition common to this disease and to scarlet fever. There is no sweating until the crisis is past, when it may be profuse, but is generally less noticeable in children than in adults.

Herpes labialis may occur with pneumonia in children, generally in mild cases, but it is less common than in adults. Schlesinger found it in 18 per cent. among 173 cases. A slight icteric tinting of the skin and conjunctiva is occasionally present, and jaundice occurs in some cases.

*Urine.*—The urine is scanty and concentrated and there is generally a diminution or even absence of chlorides, as in pneumonia of the adult. Albumin is found in a proportion of the cases, according to Schlesinger in 28 per cent.

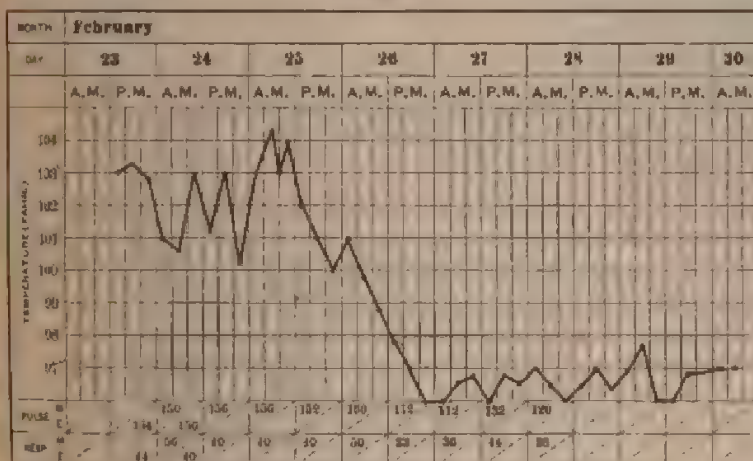
*Temperature.*—The temperature rises abruptly, so that in a few hours it registers  $104^{\circ}$  or even  $105^{\circ}$  F. At this point, or thereabouts, it remains throughout the attack, with remissions of  $1\frac{1}{2}^{\circ}$  to  $2^{\circ}$  in typical cases, though more marked remissions of  $3^{\circ}$  or  $4^{\circ}$  may occur, and are more common the younger the patient. Rarely, it swings like a hectic temperature, and this in cases which present no other peculiarities. On observing such a chart we must be sure that the oscillations are not the result of sponging to reduce fever.

*The Crisis.*—At the crisis the temperature falls rapidly from a high level to normal or subnormal; thus it may drop from  $105^{\circ}$  to  $97^{\circ}$  F. within twelve to eighteen hours. Very commonly the temperature falls to near normal on the day before the true crisis and rises again. After the crisis the temperature remains subnormal with fluctuations of less than  $2^{\circ}$  for a few days, and then becomes normal. When the crisis appears the pulse and respiration rapidly resume the ratio of



health, and the patient, from a condition of considerable distress, very quickly regains a state of comfort. The time at which the crisis appears varies considerably. The accompanying chart (Fig. 137) shows the incidence among eighty-six cases collected by myself.

FIG. 136



Temperature chart, case of lobar pneumonia.

FIG. 137

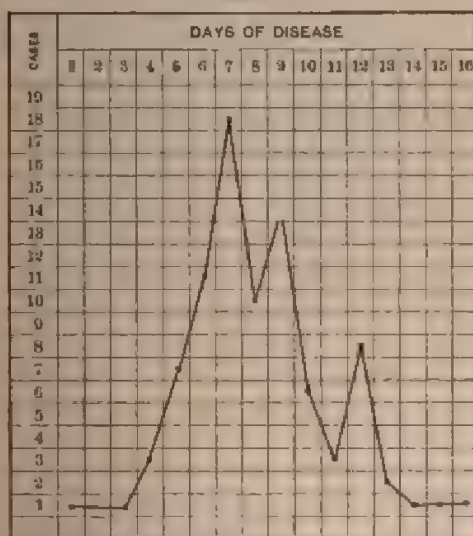


Chart showing day of crisis in eighty-six cases of lobar pneumonia.

Probably more abortive cases of pneumonia occur in children than in adults, but their prevalence is hard to estimate. In some cases the attack lasts but one or two days, the lung condition never passes beyond the stage of engorgement, and, in the absence of physical signs, the

nature of the condition often remains unrecognized. With regard to the frequency of a crisis in lobar pneumonia of children, I find, among cases collected by myself, that during the first year of life 35 per cent. ended so, and during the second year 31 per cent. Among 192 cases of all ages up to fourteen years, 86, or 45 per cent., ended by crisis, so that it appears to occur in nearly half the cases of croupous pneumonia in childhood.

**Physical Signs.**—These vary according to the stage of the disease at which the case comes under observation. At the onset nothing abnormal is found in the chest, and this must not be allowed to throw doubt upon the diagnosis, which can generally be determined on other grounds. Indeed, it generally happens that the signs do not appear for some days after the onset, and they may even be delayed until the crisis is past. This delayed appearance of physical signs has been explained on the supposition that in childhood the process often begins in the central portions of the lung and gradually spreads thence to the surface. This supposition is untenable, since consolidation, when covered by healthy lung tissue, does not give rise to the weak breathing so commonly noticed at the first sign of pneumonia, as may be proved by listening at the lung's apex in front in cases where consolidation is confined to the posterior portions. Under these circumstances is heard harsh breathing, due to the overacting lung tissue in front, and, through this, distant bronchial breathing from the solid area behind. More probably the tardy appearance of signs depends on a true delay in passing from the stage of congestion to that of definite consolidation.

The earliest sign to be found is a weakening of the breath sounds, which may progress for two or three days until breathing is almost entirely suppressed before it becomes tubular. This diminution is probably due to restricted expansion of the lung, owing to vascular engorgement. A little later a rise in tone of the percussion note may be appreciated, and, therewith, often some accentuation and lengthening of the expiratory sound, and slight increase of vocal resonance.

When consolidation is fully established the following signs are present.

**Inspection.**—If the consolidation involves a large area of lung tissue some loss of mobility on the affected side will be observed.

**Palpation and Percussion.** With the hand laid upon the chest wall this impaired movement may be clearly appreciated, and occasionally a friction rub or intrapulmonary râles may be felt. Vocal vibration is not obtainable in young children owing to the treble quality of the voice. On percussion over the consolidated area a high-pitched, impaired, or dull note is obtained, and the finger appreciates some increase of the normal resistance. This varies much according to the amount of consolidation present. As a rule, the resistance is not such as suggest the presence of fluid, but occasionally, if the consolidation extensive, the resistance and dullness may, to some extent, simulate that of effusion. When this is so, it is sometimes due to a layer of fluid outside the solid lung, but not always. Around the consolidated area the percussion note shades off gradually into that of healthy lung.

the note may be "boxy" over clear areas. Occasionally, the son note may be truly tympanic over the pneumonic lung, and more commonly, I think, when the process involves the apex (38).

*Respiration.*—The breath sounds at the very commencement are suppressed, as already pointed out; later they are harsh in quality, with prolonged expiration, and soon the expiration becomes bronchial while the inspiration remains vesicular, a condition to which the term bronchovesicular is sometimes applied. When consolidation is fully established, high-pitched, whiffy bronchial breathing, inspiratory and expiratory, is heard, often very intense and near

FIG. 123



Lobar pneumonia in a girl of fourteen months. shaded area represents the consolidated lung and the dots the crepitations.

loud, but occasionally soft and distant. It is accompanied by a series of sharp, consonating râles of medium size, occurring mostly at the end of inspiration. The fine-hair crepitation of adults is but rarely heard in childhood.

Occasionally, on listening over the consolidated area, nothing but weak breathing is noted until the child coughs or cries, when, for a short time, intense bronchial breathing and sharp râles appear, but soon give place to weak breathing. This indicates a blocking of the bronchial tubes with secretion, and in some cases of massive consolidation the breath sounds may remain suppressed for some days, giving rise to a marked dullness and increased resistance to percussion, a close resemblance to the signs of effused fluid.



Though the sharp inspiratory râles mentioned above are very characteristic, quite commonly no added sound is audible over the consolidated area, or, at most, a sharp click at the end of inspiration, and no further added sound than this may be found from first to last in some cases. Over the rest of the lungs the breath sounds are normal or perhaps somewhat exaggerated; in some cases there are, in addition, scattered, dry, bronchitic sounds. Although the pleura is invariably inflamed over the pneumonic area a definite friction rub is not often to be detected. When present it is fine in quality, and is often difficult to distinguish from the accompanying intrapulmonary sounds.

Vocal resonance is increased over the consolidated area, often intense and bronchophonic. When the process is at the base of the lung the scapular angle is generally the point of most intense conduction. In some cases increase of vocal resonance is the only discoverable sign of consolidation.

*Resolution.*—When the temperature has fallen, either by crisis or more gradually, the lung consolidation clears up with great rapidity, so that generally in three to five days the lung is either quite normal to examination or there is left only slight impairment and deficient air entry with a few râles, all of which disappear a few days later. There is no expectoration as in the adult and but little cough, the whole being removed by liquefaction and absorption. Occasionally resolution is delayed, and, rarely, it may then be incomplete and leave behind some amount of pulmonary fibrosis.

*Position of Lesion.*—In lobar pneumonia the process commonly follows the lobar arrangement of the lungs. Consequently the position of the lesion is more accurately indicated by naming the lobe, upper or lower, in which it occurs than by speaking of apex and base in this connection. When the upper lobe is affected the consolidation is mainly in front and reaches down to the third space or fourth rib; it is generally found also at the apex of the axilla, and behind involves only the suprascapular region. When the lower lobe is involved the signs are mostly or entirely behind, over the base of the chest, reaching upward perhaps nearly to the scapular spine; they extend round the side to the midaxilla at the base, but seldom farther forward, and are not rarely confined to the posterior aspect. The middle lobe on the right side is not commonly involved in lobar pneumonia.

Among 129 cases of lobar pneumonia in children of all ages analyzed by myself the following distribution occurred:

<i>Right Lung.</i>		<i>Left Lung.</i>	
Upper lobe . . . . .	40	Upper lobe . . . . .	10
Lower lobe . . . . .	21	Lower lobe . . . . .	51
Whole lung . . . . .	3		
Both lungs . . . . .		4	

Thus it appears that the left lower lobe is the most frequent seat of disease, and after this the right upper lobe which was affected four times as often as the left upper lobe. The right and left sides were affected in a nearly equal number of cases, in 59 per cent. in the lower



lobes and 41 per cent. in the upper lobes. It is thus apparent that apical pneumonia is quite common in childhood, and this is especially the case in infancy.

*Other Organs. Heart.*—In children massive consolidation of a lower lobe will generally displace the heart somewhat, and this must be borne in mind when a diagnosis between solid lung and pleural effusion has to be made. As in adults the impervious condition of the pneumonic lung throws a strain upon the pulmonary circulation, but in children this is generally well borne. The cardiac muscle is generally of good quality, and the right ventricle proportionately stronger than in later life. In fetal life the pressure in the right and left sides of the heart is equal and, hence, the muscular development of the right ventricle no less than that of the left, and this powerful right heart remains through the earlier years of childhood and is of signal value when lung consolidation occurs. When dilatation appears it often affects the heart as a whole, generally as a result of the high temperature, and the poisons of the disease. It may be considerable without giving rise to any symptoms. Any failure of the right heart is indicated by an increase of dulness beyond the normal finger's breadth (deep dulness) to the right of the sternum, and therewith an alteration in quality of the heart's first sound, perhaps a soft systolic murmur or reduplication. If the disability is great the heart's pause may be shortened, giving a rhythm like that of the fetal heart, and therewith the pulmonary second sound loses its accentuation. These are very serious signs, and will be accompanied by obvious symptoms of distress.

The *abdomen* may be distended somewhat and tympanitic. The liver and spleen are generally somewhat swollen, the latter sometimes palpable, owing to the changes brought about by the high temperature and the poisons of the disease.

*Clinical Varieties. Relapsing Pneumonia.*—Cases of relapse in lobar pneumonia are not very common, but occasionally occur. After the crisis the temperature remains subnormal for a few days, generally three to five, and then the attack is repeated and another crisis occurs. Only one relapse may occur, but occasionally three or four are seen and they generally tend to get shorter. During each attack a separate area of lung is affected, often at a distance from that formerly involved; thus in a child of two years the consolidation began in the left upper lobe, and in three relapses following the right upper lobe, the left upper lobe, and the left lower lobe were respectively attacked (Fig. 139).

*Spreading and Double Pneumonia.*—In cases where new areas of consolidation occur, whether on the same or opposite side, the outlook is rendered more serious. As a rule, the opposite lung is attacked, and not uncommonly an effusion arises on one or other side in these double cases. The duration of the attack is in any case prolonged, the crisis being delayed to the twelfth or fourteenth day.

*Abortive Pneumonia.*—This variety is more common in children than in adults. The symptoms of an ordinary onset occur, and then, often before any physical signs have appeared, a crisis occurs and the child

is rapidly well. Generally some slight changes are found in one lung, but occasionally these do not appear, and the diagnosis has to be made

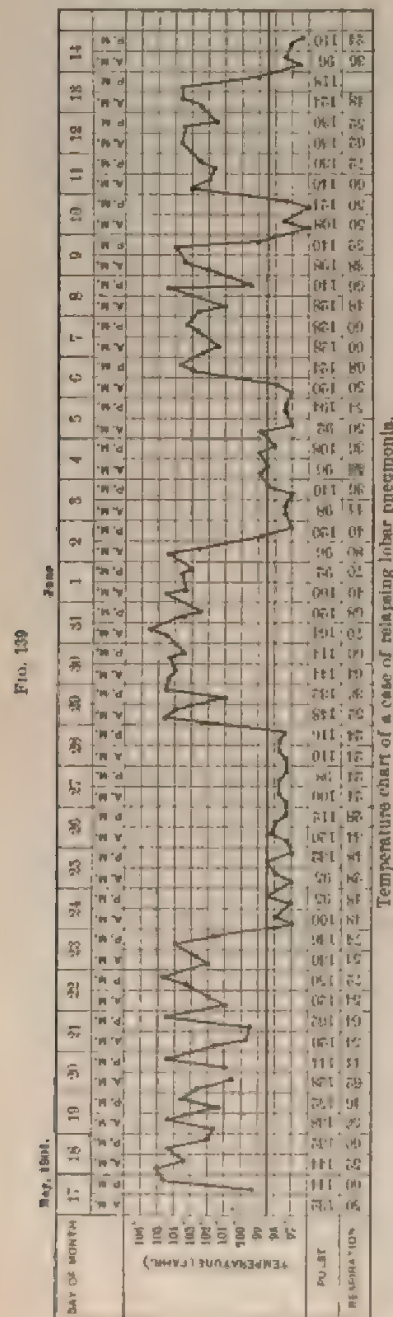
in their absence and remains conjectural only. No doubt in practice the term "febricula" covers a multitude of such cases. It is probable that the process in the lung may be arrested in the stage of engorgement and never proceed to hepatization.

*Pneumonia with effusion* will be described under complications; it might with equal justice be included under the present heading.

*Pneumonia with Cerebral Symptoms.*—These cases are very puzzling and often tempt the unwary to a diagnosis of meningitis. The symptoms are various, and include intense headache and nervous irritability, coma and head retraction, twitchings, and convulsions, either general or confined to one side. These phenomena generally occur at the beginning of the attack and disappear when the physical signs in the lung become evident, but they may last until the crisis be past. It is well in such cases to examine the tympanic membrane carefully, for not uncommonly the nervous symptoms depend upon an accompanying otitis media and only disappear when this complication is recognized and treated. Cerebral symptoms are not more common with apical than with basal consolidation.

**Complications.**—When at the expected time no crisis appears and the temperature remains high, some cause for this must be carefully sought. A spread of the disease in the lung may be responsible, but, apart from this, one of the following complications may be present.

*Teething.* Occasionally, in infants, a troublesome tooth may be the explanation. In such a case the temperature may swing and take



a very irregular course throughout the attack, its duration, besides, being unduly prolonged.

*Otitis.*—Much more commonly otitis media is the cause of the continued temperature. This is a common complication of pneumonia of all kinds in infancy, and not infrequently delays the crisis in lobar pneumonia. Sometimes, in such an event, the temperature makes wide fluctuations, but it may remain continuous, after the type of the pneumonic temperature, its persistence beyond the usual period being the only noticeable feature. Not infrequently I have observed the crisis, delayed until then, follow immediately on a successful puncture of the tympanic membrane. As a rule, the otitis runs its course with the pneumonia, but occasionally it precedes it and is then, perhaps, a causative factor in the attack, since the pneumococcus is a common cause of otitis in childhood.

*Pleurisy.*—Dry pleurisy with the formation of fibrin over the consolidated area occurs in all cases, and gives rise to no symptoms beyond the occurrence of pain during the attack and subsequent adhesion of the pleural surfaces. Occasionally the pleurisy may attain more important dimensions, and result in effusion either serous or purulent. In many cases clear fluid is poured out between the surfaces, and may remain localized by adhesions giving rise to wooden dulness and feeble breath sounds over the pneumonic area; it is ultimately absorbed and causes only a tardy resolution. When an effusion arises the crisis is often delayed until, perhaps, the twelfth or fourteenth day, and thereafter a slight temperature is maintained by the pleural inflammation for a while. If the fluid is removed with the aspirator this will often bring about a crisis which has been delayed up to that point. Dulness and perhaps pleural friction remain for a week or two after the crisis. Though the fluid is generally encapsulated it may be free in the pleural cavity, and occasionally with an apical pneumonia accumulates at the base below it. Pleural effusion is not uncommon on one or other side when the pneumonia is double.

Often the fluid drawn off is somewhat turbid, and is found microscopically to be filled with pus cells. This will naturally cause anxiety as to the outcome, whether it will clear up like a serous effusion or progress to the formation of an empyema. My experience has been that such fluid is not uncommonly absorbed, or at least the patient may escape with no more serious operation than aspiration. *Empyema* is a more serious complication to deal with and is accountable for a large number of deaths in croupous pneumonia of children. In many cases it is probably present from the outset.

*Bronchopneumonia.*—This is a common complication in the first two or three years of life and not infrequently leads to a fatal issue. In some cases there is a definite crisis and bronchopneumonia occurs subsequently, but often the temperature never falls and a case which began as a lobar pneumonia progresses as a bronchopneumonia. The process often starts in the affected lung and a centre of hepatization may sometimes be found surrounded by areas of bronchopneumonia. It is



probable that often, where a croupous pneumonia is diagnosed and only bronchopneumonia found at the autopsy, the disease was really a lobar pneumonia at the outset, and the hepatization has cleared up, leaving to view only the bronchopneumonic complication which led to death.

*Purulent Pericarditis.*—This is an occasional cause of death and either occurs by itself or more commonly in conjunction with empyema. It may be suspected in some cases, but is not very often diagnosed during life. The quantity of pus is seldom great and, in the supine or semi-supine position, gravitates to the back of the heart whose apex swims forward into contact with the chest wall. Consequently, the sounds are not muffled and the increase of cardiac dulness may be hardly appreciable. Moreover, a friction rub is very rarely present. The most valuable guide to the diagnosis of this condition is the marked general and respiratory distress which are out of all proportion to the lung condition present, and generally lead to orthopnea. Of physical signs the cardiac dulness is the most valuable. If it extends well outside the apex beat in the fifth space (in the fourth space this is of no value, as it often does so here in dilatation), if it rise to the second rib, and stretch well to the right of the sternum in the fifth space, and if there is appreciable resistance to percussion—these are signs of great value, but they are often absent.

*Purulent Peritonitis.*—When purulent peritonitis occurs as a complication it is generally only part of a widespread infection of the serous membranes, a pneumococcic polyserositis, and adds little or nothing to the symptomatology, being on this account often overlooked.

*Purulent meningitis* is of occasional occurrence, generally in association with some of the other pus complications. It is perhaps more often suspected than found owing to the occurrence of pneumonia with cerebral symptoms, but in these cases the symptoms occur early in the disease. When they are of late onset they are generally due to meningitis.

*Abscess and gangrene* of the lung are rare complications and are described under separate headings.

*Sequels.* These are usually not of a serious nature and often only temporary in character. Bradycardia often occurs in older children, the pulse dropping to 60 or less and recovering itself again during convalescence. Some amount of cardiac asthenia may be left in anemic and weakly children but seldom gives much trouble. *Pulmonary fibrosis* is a rare sequel to lobar pneumonia and is probably the most serious that may occur; it is, however, far less common than after bronchopneumonia. *Recurrence* of pneumonia is not uncommon; among hospital cases in older children a history of previous attacks can be obtained in a certain proportion. Sometimes a great many recurrences occur. Cases of pulmonary fibrosis with recurrent acute attacks must not be mistaken for or included among these.

*Diagnosis.* This, in young children, is sometimes a very difficult matter. At the onset the most striking resemblance exists to scarlet



fever; when consolidation has appeared two other pulmonary diseases have to be considered. On the one hand are cases of bronchopneumonia with lobar consolidation; on the other pleural effusions whose signs are sometimes indistinguishable (I speak advisedly) from those of solid lung.

*Scarlet Fever.*—The onset of pneumonia often resembles that of scarlet fever very closely. Vomiting, headache and fever, with a dry, burning skin, are common initial symptoms in both, and many points require consideration before a conclusion can be arrived at. The tongue is furred in both conditions, but the raw, red tip and sides and the swollen papillæ in scarlet fever, especially when taken with the dry, red lips, may be of assistance. Sore throat is strong evidence in favor of scarlet fever. The dry, pungent skin belongs to both scarlet fever and pneumonia, and it is this, in many cases, which leads to doubt. A passing erythema may be seen with pneumonia, but not, of course, the persistent punctiform rash of scarlet fever. It is generally before the eruptive period that doubt exists. Much the most important diagnostic point is one on the side of pneumonia, namely symptoms pointing to the respiratory system. Rapid respirations accompany high fever of any causation, but the disturbed pulse-respiration ratio with expiratory grunt, working alæ nasi, and often some cough, point to pneumonia. In scarlet fever it is the pulse that is especially rapid; in pneumonia, the respiration.

*Bronchopneumonia.*—Cases of primary bronchopneumonia often present a sudden onset closely similar to lobar pneumonia; secondary bronchopneumonia is not likely to lead to mistake, as it is preceded by bronchitis of the larger tubes and often follows one of the exanthemata. In a case, then, with sudden onset in a young child the question of diagnosis may arise, and it must be borne in mind that bronchopneumonia is uncommon after the age of three years. If the lung signs are those of a scattered bronchitis with patches of consolidation at both bases behind, a diagnosis of bronchopneumonia will be readily arrived at; if consolidation is confined to one apex, on the other hand, lobar pneumonia may be accepted with great certainty. These are the extremes which leave little room for doubt, but if there is marked consolidation at one base many nicer points of distinction require consideration.

Inspection, palpation, and percussion give no help. The character of the breath sounds, moreover, offers no distinction, but if they are unaccompanied by râles it is almost certainly a lobar pneumonia, and if the râles are few in number it probably is; on the other hand, if they are abundant and spread beyond the limits of the consolidated area, it is more probably a bronchopneumonia. Râles elsewhere over the chest must be looked upon with suspicion, though they may occur with lobar pneumonia, and in all cases of doubt the appearance of sharp râles or consolidation elsewhere, especially at the opposite base, must be looked for to confirm a suspicion of bronchopneumonia. If the nature of the consolidation has remained doubtful, a crisis is in favor

of lobar pneumonia, though it appears, on rare occasions, at the termination of a bronchopneumonia; when it does so the temperature more often shows some subsequent fluctuations than the subnormal course seen in lobar pneumonia. After the attack the course of resolution is often a useful confirmatory sign—in lobar pneumonia the lung often clears in three to five days; in bronchopneumonia it is never clear by the end of a week, and it generally takes ten days or longer before all signs are gone.

The temperature generally fluctuates in bronchopneumonia; in lobar pneumonia it is often continuous, moreover, it takes a generally higher level in the latter disease, a temperature of  $104^{\circ}$  or  $105^{\circ}$  F. being common and very suggestive. Hyperpyrexia, if this occurs, is still strong evidence on the side of lobar pneumonia. Cough is often distressing in bronchopneumonia; it is generally slighter and may be absent in lobar pneumonia. Constipation is common in lobar pneumonia, but very rarely found in bronchopneumonia; diarrhea may occur with either, but more commonly with the latter. It must not be forgotten that, though the victims of the secondary bronchopneumonias are generally poorly nourished and sickly children, primary bronchopneumonia, like lobar pneumonia, commonly attacks the healthy.

*Child and Adult.*—Lobar pneumonia in the child is closely similar to the same disease in the adult, but certain important points of difference need emphasis. At the onset a *rigor* is usual in the adult, rare in the child; *vomiting* is usual in the child, rare in the adult. Cough is a more important feature in later life and is accompanied by the characteristic rusty sputum; in children cough is often slight or even absent, and there is no expectoration.

The *crisis* in childhood is a somewhat less important feature than in adult life; it occurs in a smaller proportion of cases, the fall of temperature is often less rapid, and is accompanied by less sweating. *Hyperpyrexia* occurs less commonly in children.

The pulse-respiration rate is disturbed in children as in adults, but as a diagnostic sign it is of less value, since in childhood it is readily disturbed by lung conditions other than pneumonia, and must be considered in conjunction with other signs.

*Apical pneumonia* is common in childhood and occurs in about 10 per cent. of all cases; in adult life it is far less often found. *Abortive* cases are occasionally met with in the adult, but they are probably far commoner in childhood, though at either age it is impossible to estimate in what percentage they occur.

The difference in the *mortality* of the disease in childhood and later life will be described in the next paragraph.

**Prognosis.** This depends in the first place on the age of the patient, for the mortality is very much higher in infancy than in the later years of childhood. Among 196 cases analyzed by myself the mortality was 25 per cent. in the first year of life and 15.4 per cent. below the age of two years. Infants die from mere stress of the disease itself; in older children recovery takes place unless some fatal complication supervenes.

Of complications, the infection of serous cavities by the pneumococcus is the most serious; in some cases many serous cavities are simultaneously affected and the condition was probably a pneumococcus pyemia from the beginning, the pneumonia being only an accidental accompaniment. The mortality for children above the age of two years among my cases was only 2.3 per cent.; that for children of all ages 6.6 per cent. We thus see that lobar pneumonia has a low mortality in childhood compared to the 20 per cent. of adult life, and that in older children the prognosis is extremely good. It is largely for this reason that a diagnosis from bronchopneumonia is important, since the ultimate outlook is so different in the two conditions.

**Treatment.**—If the case is seen at the very onset a hot bath should be immediately given, the child thereafter being placed between blankets, and a diaphoretic hot drink containing acetate or citrate of ammonia administered. The disease will occasionally abort at the very beginning, though it is very doubtful whether the remedies employed are in any way responsible for this happy termination. The benefit of the doubt should be given on the side of active treatment.

When the disease is fully established it is well to remind one's self at once of its self-limited course and the non-existence of any specific treatment. Our attention must be given to watching the patient through the attack, usually short and of favorable termination in the child, and dealing with the complications as they arise.

The child must be put to bed, and covered with light clothing in a well-ventilated room. A milk diet must be given at regular intervals, and in moderate quantities; the illness promises to be short and it is wise not to overtax the stomach. Cold water should be freely given to relieve thirst, but in small quantities at a time. The bowels are generally confined in this form of pneumonia and it is as well to relieve them at the beginning. Calomel, 0.065 gm. (1 gr.), with 0.13 to 0.2 gm. (2 or 3 gr.) of powdered scammony may be given for this purpose. Too violent purgation is to be avoided. In a case of normal course drug treatment and the administration of stimulants are needless and even harmful, but the skin discomfort may be somewhat eased, and no harm done by small doses of the citrate or acetate of ammonia, 1 c.c. (15 minims) of the liquor for a child of one year, at three or four hourly intervals.

The skin should be sponged over with warm water two or three times daily without disturbing the child to reach inaccessible parts, and if the temperature is high some means should be taken to keep it within reasonable limits. Children are generally less distressed by high temperature than are adults, but it must be remembered that its effect on the tissues is deleterious even though it is well borne by the patient, and that especially where the remissions are but slight. If the remissions are considerable and the child not distressed a temperature of 104° F. may be left alone, beyond the daily spongings; if the temperature is continuous the sponging should be more assiduously performed. Cold bathing as usually done distresses the child, and may not be followed by a healthy reaction.

With the higher temperatures, which are not at all uncommon with pneumonia in young children, the fever should be controlled by cold packing or by "cradling" or "ice-cradling." In the former method the child is wrapped in a towel wrung out of cold water and a blanket laid over. The pack is changed at intervals until the temperature is sufficiently reduced. "Cradling" is a better method, as it involves no disturbance of the patient. The bed-clothes, which should be light, are raised over a wicker or metal "cradle," which crosses the patient from side to side like a wide bridge. The "cradle" is open at the end for the air to enter, the child's body being covered with a night-dress or thin blanket. In "ice-cradling" bags of ice are hung along the top of the cradle at intervals. The child may remain "cradled" for days, or as long as the temperature requires it. In all these methods it is of the utmost importance that the feet and legs be well clothed and kept warm with hot-water bottles. This is so often overlooked that physicians and nurses must be reminded of it from time to time. Antipyretic drugs are but rarely necessary or advisable. When cold is used to reduce fever the temperature falls several degrees after the agent is removed, so it is well to stop the application when 102° F. or thereabouts is registered.

Nervous symptoms, as delirium and sleeplessness, are generally controlled by reducing fever, but occasionally other means are needed in addition. For either small doses of Dover's powder may be tried, and if occurring late in the illness they may be due to exhaustion and be removable by alcohol and stimulants.

The heart is less often a cause of trouble than in pneumonia of adults; nevertheless its condition must be carefully watched. Failure may occur in either of two ways: first, the whole heart may be poisoned by the fever and products of the disease, in which case the child is pallid, with cold extremities, rapid, weak, and irregular pulse, and perhaps vomiting; or, second, the disability may be of the asphyxial type, involving the right chambers mainly, and causing cyanosis and increased dyspnea, with widening of the cardiac dulness to the right of the sternum. For both conditions cardiac stimulants must be given, and for the latter, in addition, in England we depend on bleeding by the application of leeches over the chest wall or liver. In this latter class of cases nitroglycerin is regarded in America as useful in relieving engorgement of the right heart.

Oxygen is to be administered in both these classes of cases.

The best stimulant is strychnine, which may be given in doses of 0.0003 gm. ( $\frac{1}{3000}$  gr.), at four to six hourly intervals for a child of one year. Alcohol is preferred by some and is especially useful for heart weakness of the syncopal type. It is best given in the form of whiskey or brandy, of which  $\frac{1}{2}$  to 1 ounce may be given daily to a child of one year. This may be exceeded for short periods if necessary, and it is always well to reserve the largest proportion of the daily allowance for those times (generally the small hours of the morning) when the temperature and vitality are lowest.

If brandy or whiskey is objected to rectified spirit may be tried in



half the dose, the taste being hardly perceptible if given in milk. Caffeine and digitalis are recommended by many. I have had no experience of their use. Jacobi mentions camphor and musk, and it is reasonable to believe that they may be of benefit.

Pain in the side may be relieved by strapping, if well borne, or by a mustard plaster, or light poultice of mustard and linseed (1 to 5 or 6). Opiates may be given if necessary.

Throughout the attack a watchful eye must be kept for complications, especially those more treatable ones, otitis media and pleural effusion.

## CHAPTER XXVI.

### PLEURISY—EMPHYEMA—PNEUMOTHORAX.

#### PLEURISY.

INFLAMMATION of the pleura leads to one of three results, either dry pleurisy, in which no measurable quantity of fluid is formed, or sero-fibrinous pleurisy, or empyema. The first is generally an accidental complication of some pulmonary inflammation, either a pneumonia or a tuberculous consolidation; the second is generally, to all intents and purposes, a primary disease and is commonly caused by the tubercle bacillus, though it may accompany croupous pneumonia; the third may be primary, but is often secondary to a lung inflammation, either pneumonia, in which case the pneumococcus is generally the cause, or some septic lesion, as an infarct or abscess, due to the inroad of some one or other of the pyogenic organisms.

**Dry Pleurisy.**—Dry pleurisy may be dismissed in a few words. It accompanies any lung inflammation which reaches the surface and is found in lobar pneumonia, in bronchopneumonia where the consolidation is considerable, over infarcts, and in chronic tuberculosis. The surface appears dry and rough and generally covered with a thin layer of fibrin, and the condition gives rise to the friction rub so frequently heard over consolidated areas. It leads to permanent adhesions between the pleural surfaces and, judging from the frequency with which these are found in the postmortem-room, this and the following form must occur with considerable frequency.

**Sero-fibrinous Pleurisy. Etiology.**—It may occur as a localized collection of serum over the surface of the solid lung in croupous pneumonia, and in this connection is described under the heading of that disease.

The disease to which the name pleurisy is usually given is a primary inflammation of the pleura, and occurs nearly always in children above the age of two years. It is probably of tuberculous nature in nearly all cases, though the tubercle bacillus cannot always be demonstrated; it is, however, significant that the more perfect the methods employed the larger is the proportion in which this organism is isolated and by a recent method, in which the congealed serum was liquefied by digestive agents, the tubercle bacillus was found in all the cases examined. Moreover, the fluid is practically always sterile on culture media which at any rate, excludes other organisms. The majority are probably primary, just as tuberculous peritonitis may be primary, but it seems likely, on the other hand, that in some cases the process spreads from caseous lymph nodes lying beneath the pleura between the pulmonary

lobes, these in their turn having been infected by spread from that common primary focus, the lymph node or nodes beneath the bifurcation of the trachea. It is a significant fact that delicate children are so often attacked with serous pleurisy, and that so commonly a long history of cough and wasting, often following measles, precedes the onset of the pleural inflammation.

Certain influences, such as cold, which tend to lower resistance, may be the factors immediately determining the onset, and among these injury, such as a blow on the chest, must be included.

Serofibrinous pleurisy is occasionally rheumatic in origin, but I have never seen a large effusion due to this cause.

**Pathology.**—The fluid is clear, greenish yellow in color, and soon forms a translucent clot on standing. The pleural surface is covered with a layer of fibrin, which is generally thin, and most marked on the visceral pleura, but may form a thick, greenish layer. Where thick, it generally shows a shaggy surface, but where thin may have a beautiful reticulated appearance. The lung is partially or entirely collapsed, the surface looking white and thickened on this account; it is tough to cut, heavy and airless, and the cut surface is of a dark chocolate-brown color crossed by the obvious double white lines of the bronchial tubes. The neighboring organs are variously displaced according to the amount and position of the fluid.

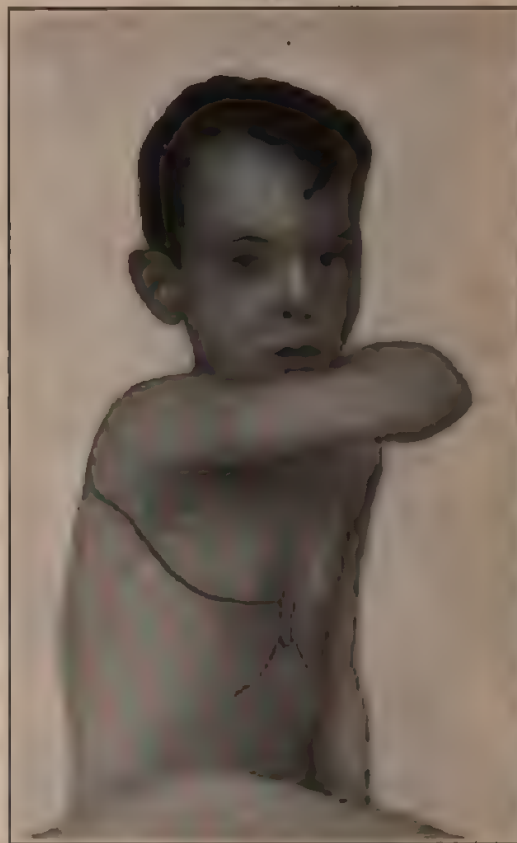
**Symptomatology.** *Onset.*—The onset is seldom abrupt; generally the symptoms develop themselves gradually over a period of several days. Vomiting is rare as an initial symptom; *chilliness* occurs in a fair proportion of cases, but does not amount to a rigor. It is accompanied by *cough*, *fever*, and *pain* in the side. Frontal *headache* is complained of, and the bowels are nearly always constipated. The breathing becomes difficult, quick, and shallow, as more and more fluid is effused; the effusion generally forms rapidly. The child is listless and quiet, but is even not sufficiently ill to take to his bed, and may go about for several days before the importance of the condition is recognized. Indeed, the onset is occasionally so insidious that dyspnea caused by a large effusion is the first thing noticed.

Very commonly a history of cough and wasting of some months' duration preceding the immediate onset is obtained, as already mentioned, and I have observed that measles is a very common antecedent. The *cough* is sometimes the first symptom noticed but often it is delayed for a day or two; it is short, dry, and hacking, and unaccompanied by expectoration; it generally causes pain in the side or accentuates that already present. The *pain* is of early appearance, and is located either in the side of the chest or, not infrequently, in the abdomen, sometimes simulating that of appendicitis or some other abdominal disease, and on one occasion I have seen it referred to the region of the shoulder-joint on the affected side. The pain is generally severe and increasing and accompanies inspiration, especially if a deep breath is taken; movement of the chest on the affected side is often voluntarily avoided to avoid it. At the beginning the pain is increased by pressure

over the inflamed area, and the child inclines to lie on the back opposite side, but as the effusion forms the pain goes, and it lies on the affected side to give more play to the healthy lung.

The *breathing* depends on the local conditions present. beginning it is quick and shallow, owing to pain caused by deflation; when an effusion is present, if small, the breathing may be appreciably altered from the normal, but when large the respiration

FIG. 140



Pleurisy: line of fluid anteriorly.

are again rapid and shallow, the alae nasi work, and the pulse-ratio may be altered so as almost to simulate that of pneumonia.

The *temperature* is generally high during the acute stage,  $102^{\circ}$  F. being a common figure. It usually remains raised for five to a week and sometimes longer, with considerable fluctuation. The *skin* is hot and often dry during the day, with profuse sweating at night and early morning hours.

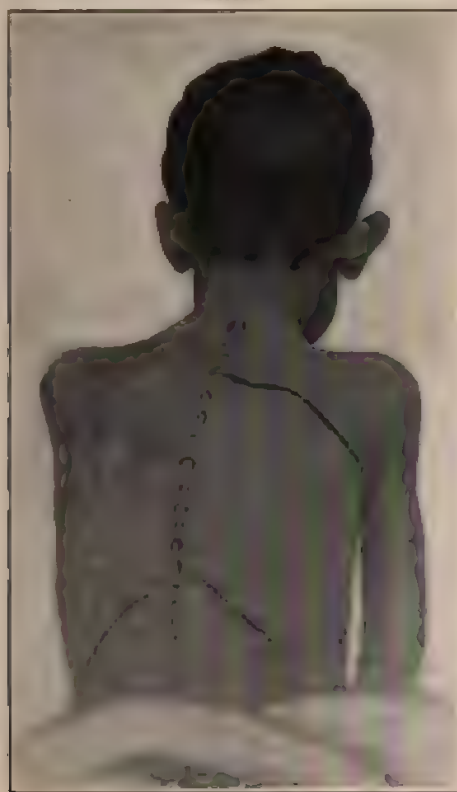
During the attack the child loses flesh, is weary and restless, constipation often demands treatment. The tongue is furred,



with often a slight straw-yellow tint observable, mostly around the mouth. This is less noticeable in cases of serous effusion than in pyæmia, where the rapidly produced anemia heightens the effect. Finger-tips may become glazed in a few cases where the fluid has not been absorbed for two or three weeks, but any definite "clubbing" occurs with uncomplicated serous effusion.

*Location of Lesion.*—Serous effusions are seldom loculated, but may sometimes. When they occur over the solid lung in croupous pneumonia they often remain localized and do not spread around the chest.

FIG. 141



Pleurisy: line of fluid posteriorly.

Serous effusions may be loculated at the commencement, but if they increase in size they become free and spread around the base of the lung so that, as a rule, the signs of fluid are found both in front and behind. In children an interesting form of loculated serous effusion sometimes occurs and may readily lead to a false diagnosis. This is a collection appearing in the front of the chest over the middle lobe on the right side, giving rise to signs which are liable to be mistaken for a localized enlargement or pulmonary collapse. The collection may be absorbed without further spread, but generally it increases in size, its

limitations are broken down, and the fluid spreads around the chest in the ordinary manner.

Commonly, a serous effusion is found at the base of one or other pleural sac, but occasionally it may be double, both pleura being involved, and if the amount of fluid poured out is moderate, the case may run as favorable a course as when the effusion is single. Nevertheless, in such cases the probability of some primary tuberculous lesion elsewhere being the cause of the trouble must be taken into consideration.

The quantity of fluid varies between an ounce or two and as much as one and a half pints in a child of three and one-half to four years of age, the larger effusions being distinctly less common.

**Physical Signs.** *Inspection.*—On examining the chest it will generally be observed that movement is deficient on one side, though with effusions of moderate size in children this may be quite unnoticeable; with large effusions the loss of mobility is always obvious. Some enlargement of the side due to release of the elastic expansion of the thorax may be found on measurement, but can hardly be seen. The displacement of the heart's apex may be observed on inspection but is best determined by palpation.

*Palpation and Percussion.*—With a right-sided effusion the heart and mediastinum are drawn to the left by release of the elastic tension of the healthy lung, both the apex beat and the left area of cardiac dulness being found outside their normal position. In cases of large effusion the left limit may be the anterior axillary line. When the effusion is on the left side the heart and mediastinum move to the right, the point of maximum impulse is often in the epigastrium or even in the fourth space to the right of the sternum, and the cardiac dulness may reach to the right nipple line. The liver or spleen are displaced downward with large effusions.

Vocal vibration cannot be obtained in children owing to the treble quality of the voice, and thus a valuable sign of fluid is lost. A fluid wave may, however, be obtained in some cases by placing a hand on the back of the chest while an assistant percusses firmly over the front.

On *percussion* over the affected area a dull, wooden note is elicited and a sensation of great resistance is imparted to the fingers; if a considerable amount of fluid is present, the quality of this resistance is almost pathognomonic of its cause, only one other condition, pulmonary fibrosis, giving a comparable sensation. The dulness may extend to the apex in large effusions, but is always most absolute at the base. When the effusion is large it will be found to cross the middle line of the chest above the heart in front, giving dulness to the opposite border of the sternum or even beyond this. In smaller effusions the upper limit is not sharply defined, but an area of impairment exists above it. Often the lung above the effusion gives a high-pitched, boxy, or even tympanic note to percussion.

Very commonly the signs of a pleural effusion reach to the scapular angle or just above this behind, to an equal height in the axilla, and often somewhat lower in front. The upper limit changes somewhat

with changes of attitude. On the left side it must be remembered that the pleural sac extends some inches below the base of the lung in the front of the chest, and that; consequently, a free effusion reaches downward nearly to the costal margin, covering the so-called "Traube's space," where stomach resonance is usually obtained. Here the fluid forms but a thin layer, and the stomach resonance tends to mask its presence, which is often best recognized by the sense of increased resistance to light percussion.

**Auscultation.**—Over the dull area the breath sounds are distant, especially at the base, where with large effusions they may be inaudible. The quality of the breath sounds varies in different cases; in older children they are often vesicular, as in the adult, but often, especially in early years, the breath sounds are bronchial or sometimes bronchovesicular. This may cause exceptional difficulty in diagnosis between effusions and solid lung in infancy. The bronchial quality of the breath sound depends, no doubt, on the condition of the lung beneath, though it is difficult to understand why the same condition should not be found in adults as in children in this respect. Perhaps the readiness with which pulmonary collapse occurs in childhood, together with the relatively large size of the bronchial tubes, may afford the explanation; in empyema it depends in many cases on inflammatory consolidation of the underlying lung.

Pleural friction may be audible at the upper limit of the effusion or will appear when absorption takes place. In childhood it is very fine in quality and difficult to distinguish from intrapulmonary râles; it generally accompanies both inspiration and expiration. In cases where it is loud and of sharp, resonant quality, the sound may often be conducted to the opposite side of the chest, giving rise to errors of diagnosis unless the possibility of this is recognized. The fact that the quality of the sounds on the two sides is identical, the one being but a faint replica of the other, will generally point to the true condition present. In some cases both sounds may be simultaneously arrested by turning the patient on to the affected side and thus limiting the movements of the rubbing surfaces.

The vocal resonance is diminished over the effusion. As in adults, it often has a peculiar nasal quality which may be very marked, giving a bleating tone to the voice (egophony). This is usually found in cases where the breath sounds are bronchial and is most marked at one spot, generally the scapular angle. Above the level of the effusion the vocal resonance may be somewhat increased.

**Termination.**—After remaining in *statu quo* for a variable period, often but three or four days after it has attained its maximum, the effusion begins to be absorbed, and is generally gone or nearly gone by the end of the second week, though it may remain longer. The rest for the inflamed surface afforded by the fluid covering has allowed healing to take place, and the lesion is cured. Slight impairment at the base and weak air entry, due to a sodden and partly collapsed condition of lung, remain for a short time after the fluid has disappeared.

**Diagnosis.**—The diagnosis of serous effusion seldom presents the same difficulties as does that of empyema. The subjects are generally old and the fluid is nearly always free in the pleural cavity. It is by loculated effusions, so often found in empyema, that the signs of pneumonia may be closely simulated, hence the diagnosis between these conditions must be considered when empyema is dealt with. The diagnosis from pulmonary collapse has been already considered under that heading.

**Prognosis.** Serofibrinous effusion is never fatal on its own account with the exception of certain rare cases of sudden death where the displacement of neighboring organs has been considerable. As a rule the condition clears up, and nothing further may happen, but it is well to remember that double effusion often implies a pre-existing tuberculous focus, and that even primary cases are generally tuberculous in nature and may be followed by pulmonary tuberculosis.

**Treatment.**—At the beginning the patient should be put to bed, the bowels opened with a calomel purge and their subsequent regular action attended to. The diet should be light, a diaphoretic mixture may be given, and the pleural pain treated by a firm bandage or, if required, by a hypodermic injection of morphine, 0.003 to 0.005 g ( $\frac{1}{20}$  to  $\frac{1}{12}$  gr) for a child of four to five years. Leeches or cupping may often afford relief. In the majority of cases the temperature falls within a week, and the fluid begins to be absorbed after about ten days, often disappearing by the twelfth or fourteenth day. In cases of effusion so large that the whole pleural cavity appears filled, with considerable displacement of the surrounding viscera, it is well to aspirate a part of the effusion at once. When the collection is smaller it may be removed in two and a half to three weeks, and iodides in the form of potassium iodide, 0.3 gm. (5 gr.) for a child of four or five years, may be administered while in a diuretic mixture containing 0.13 to 0.2 gm. (2 or 3 gr.) of potassium nitrate with infusum scopolii, 8 c.c. (2 dr.). A robust but copious diet should be given, the bowels kept freely moved, and the chest wall at the same time stimulated with local applications of iodine paint.

If at the end of this time there is no change, the fluid should be aspirated, as the lung is apt to expand imperfectly if left too long in a collapsed condition.

For *aspiration* the child sits forward with the arms extended over a large pillow on his knees, the chest wall is carefully prepared as for other surgical procedures, and the needle to be used is boiled. A spot at the base of the thorax behind is chosen, where the dulness is most complete and the intercostal spaces accessible, the liver being avoided on the right side. The needle is entered swiftly and steadily close to the upper border of a rib, care being taken to avoid any stabbing movement, which gives so unpleasant a sensation to the patient. The needle is in connection either with an aspirating bottle exhausted by an air pump, or with a length of tubing for siphonage. Either acts well, though the former is more efficient in case the fluid cannot be got to flow readily. The whole of the fluid may be aspirated, though there is no advantage



in removing it completely, since any that remains behind is likely to be rapidly absorbed. Accidents during aspiration are very rare indeed. If fresh blood comes with the fluid, it is well to stop the aspiration at once. Edema of the lungs occurs in rare cases, but is almost never fatal. Cases of sudden death during aspiration have been recorded, but they are very rare and have generally been attributable to the too rapid removal of a large effusion. The re-accumulation of an effusion is not of common occurrence in childhood.

Having cured the effusion, the general condition of the patient must be brought up to a high standard on the suspicion of underlying tuberculosis. Change of air to a dry, bracing climate is desirable, as also good, but judicious feeding, and some preparation of cod-liver oil with iodide or phosphate of iron. The standard of good health thus attained must not be allowed to wane.

### EMPHYEMA.

A true serous effusion rarely becomes purulent; the cause of the two conditions is different, on the one hand the tubercle bacillus, on the other hand, in children, the pneumococcus in the majority of cases. It is true that in early stages of pneumococcic inflammation the fluid may appear to be serous and will clot, but it has nearly always some turbidity due to pus cells, and grows the pneumococcus on an agar medium where the culture from the true serous effusion remains sterile. Such an effusion complicating a pneumonia may occasionally be absorbed, but when the pneumococcus is found the formation of a purulent collection may be expected.

**Etiology.**—Empyema is either primary or secondary, though the proportion of cases assignable to either of these divisions is impossible to determine. The primary cases form the smaller number and are probably always pneumococcic. The disease starts in the pleura, and the onset is either rapid, when pneumonia is simulated, or gradual like the onset of serofibrinous pleurisy. The secondary cases are generally dependent on lung diseases. The majority of them follow pneumonia, generally croupous, but sometimes bronchopneumonia, and the onset is that of one of these diseases, the pleurisy developing during its course. In some cases the lung and the pleura are attacked simultaneously, in others there is a variable interval, but usually the empyema follows very closely on the lung consolidation. Some cases, few in number, develop in the course of a pulmonary tuberculosis; some complicate fibrosis of the lung. In either case the pneumococcus is found in the pus, but in a few of the tuberculous cases the tubercle bacillus is also present. The remainder of the secondary cases are septic in origin, and follow such diseases as osteomyelitis, pyemia, appendicitis, tonsillitis, retropharyngeal abscess, or the infective fever, when the organism associated with the original disease will generally be found in the pleural pus. Septic infection of the lung is the mode by which the pleural cavity becomes infected in some of these cases.

Empyema is an uncommon disease among the well-to-do.

**Pathology. Bacteriology.**—This differs considerably in children and adults owing to the important role played by the pneumococcus in the infective inflammation of childhood. Among 77 cases in which the pus was examined by myself the following organisms were found:

Pneumococcus (alone)	66 or 85.7 per cent
Streptococcus (alone)	3 " 4.0 "
Staphylococcus aureus (alone)	3 " 4.0 "
Pneumococcus and streptococcus	3 " 4.0 "
Pneumococcus, staphylococcus, and a bacillus	2 " 2.5 "
	77 " 100.2 "

If these results are compared with the conditions found in the adult as given by Netter, the contrast is striking.

Pneumococcus	17.2 per cent.
Streptococcus	53.0 "
Staphylococcus	1.2 "
Pneumococcus and streptococcus	2.5 "
Tubercle bacillus	25.0 "

Thus it appears that in the adult the bulk of the cases are streptococic, and next to this comes the tubercle bacillus as a cause, the condition being often secondary to phthisis. In children the tubercle bacillus is an uncommon cause, and when found is generally associated with the pneumococcus; in many empyemas associated with tubercle the pneumococcus only is present.

**Morbid Anatomy.** On opening the thorax the heart and mediastinum are found displaced to one or other side if the effusion is large and untreated. Not uncommonly there is some mediastinal cellulitis, and the sternum is found to be more closely bound to the underlying structures than normally. On the affected side adhesion of the pleural surface is generally the first thing noticed, and, on separating these, the cavity of the empyema is exposed to view. The pus is generally green color, and the pleural surfaces are coated with a layer of thick, greenish fibrino-pus. The pus may be thin and watery, or it may be thick and oily in consistence, or there may exist only layers of firm, green fibrin. When the last condition is present the cause is generally the pneumococcus, but not invariably; now and then this thick, viscid secretion found in streptococic and staphylococic infections. Thin, watery pus separating into layers is suggestive of septic infection, but it sometimes occurs also where the pneumococcus only is present, and when found after death generally indicates a recent infection. Had the patient lived longer the pus would probably have become thicker.

The empyema cavity is generally bound by adhesions at certain points, even where not definitely localized. It may be found in any part of the chest, generally at the base and behind, and may be doubtless occasionally multiple pleural abscesses are found. Such cases are generally fatal, and constitute rather a pathological curiosity than a clinical type.

**The Lung.**—Pulmonary consolidation is so common an accompaniment of empyema that some amount of this is nearly always to be

found after death. Lobar pneumonia is a common cause of empyema, but is not often present at the autopsy, having generally cleared up before the fatal termination. Most commonly bronchopneumonia is found, either on the affected side or sometimes on both; it is generally a complication, and one that leads in many cases to death. It is often extensive, and may cause consolidation of the whole or the greater part of a lung; it is present in about one-half of the fatal cases. In a percentage of cases no lung consolidation is found after death, the pulmonary tissue beneath the empyema being collapsed, or partly collapsed, and often engorged or edematous. In cases of double empyema there may be bronchopneumonia on one side, and collapse on the other, and yet the signs may be similar on the two sides namely, those of consolidation. On the other hand, where the lung is collapsed beneath an empyema, the signs are often those associated with the presence of fluid in the adult, or the breath sounds may be harsh, but not distinctly bronchial.

In a small percentage of fatal cases of empyema tuberculosis of the lungs is found, and this is either of older date than the pleural inflammation, or, more commonly, is of subsequent development. In staphylococcal empyemata, and those due to other septic organisms, pulmonary infarcts or abscesses are not uncommonly found, these having led to secondary infection of the pleura.

**Symptomatology.**—The *onset* is sometimes rapid, sometimes gradual, though more commonly the former. Among cases of rapid onset a certain number start with lobar pneumonia, but it is very difficult to decide what proportion. A sudden onset does not necessarily point to pneumonia, for a primary empyema may undoubtedly begin abruptly; moreover, the crisis which occurs in many of these cases merely argues a self-limited blood infection, it is no indication of the presence of a local lung inflammation. In some cases, no doubt, the pneumonia and pleural inflammation start simultaneously.

In cases of gradual onset the symptoms are similar to those seen in serofibrinous pleurisy. There is usually a dry, hacking cough during some weeks, and the child is listless, loses the power of walking if lately acquired, wastes somewhat, and may sweat profusely at night. At the end of this time the breathing becomes short owing to the presence of effusion, and the signs of such are discovered in the chest.

When the onset is rapid it may begin with the symptoms of pneumonia (either lobar or primary bronchopneumonia), vomiting, fever, and short breath, the cough developing somewhat later, and not forming a noticeable feature. In acute cases, where the pleural inflammation is presumably primary, the onset is generally with cough (often a more prominent feature than in pneumonia), together with fever and pain in the side, which may in some cases be so severe as to cause screaming. The dyspnea is not such a marked feature as with pneumonia, and may not be noticeable unless a large effusion is present. The child is restless, at night, loses appetite, is costive, and rapidly wastes, and becomes intensely anemic if the disease remains untreated.

The *cough* is dry, hacking, and ineffectual, and unaccompanied by any expectoration. It often causes pain or increases that already present. The *pain* is usually in the side of the chest, but it may be referred to the abdomen, or to the cardiac area when on the left side. It is sharp and stabbing in character, and often produces an expression of great distress, the child crying out or screaming at intervals. Pains all over the body are sometimes complained of at the onset.

*Dyspnea*.—When the condition begins with pneumonia the respirations take the type seen in that disease, the pulse-respiration ratio being generally much disturbed; after the crisis, if such occurs, the respirations may fall to normal if the amount of effusion is small. In cases of primary pleural inflammation, whether of sudden or gradual onset, the amount of respiratory disturbance depends mainly on the quantity of fluid poured out. When the effusion is small, there is little disturbance; when the effusion is large the respirations may be rapid, the disturbance of the pulse-respiration ratio reaching 3 to 1, rarely more.

*Fever*.—The temperature may be like that of pneumonia at the onset and there may be a crisis, either delayed or at the normal time, after which the temperature makes slight rises until the empyema is treated. It is surprising how slight an increase of temperature often accompanies the presence of an empyema. At its onset there is usually a high temperature, often with marked fluctuations, but when once established septic absorption seems to be slight in many cases and the temperature often remains between 99° or 100° F. at night, and normal or somewhat subnormal in the morning, the fluctuations being often but 1° or 1½°. It is important to recognize the fact that a low temperature is not incompatible with the presence of an empyema. In many cases a temperature of 101° F. with somewhat large fluctuations precedes operation, and a drop to normal or subnormal follows it with no subsequent rise. Occasionally the temperature is low before the operation and becomes high and swinging for several days after it, the disturbance having led to greater septic absorption, especially in cases where drainage is imperfect. Taking it altogether the temperature in empyema is very variable and erratic, and not in any way distinctive of the condition causing it. As an indication of septic absorption it is, however, of value, and will draw attention to deficiency of drainage during treatment.

*Skin*.—Night-sweating may occur in empyema, though the skin is harsh and dry throughout the day. Where a patient with lobar pneumonia sweats profusely all is not normal, and empyema may cause this in some cases where the two diseases are associated.

In pleurisy of long duration a peculiar straw-yellow tinting of the skin is to be observed in the pale regions of the face, round the eyes and mouth especially. In cases of empyema this is much more marked and may be seen on the body as well. It is associated, in empyema, with intense anemia and the peculiarly flabby and wasted muscles which are found when the condition has remained untreated for some



weeks. This yellow, anemic skin is valuable in diagnosis, though a somewhat similar appearance in children is noted with purulent affections elsewhere, as in infective meningitis or pericarditis. Clubbing of the finger-tips occurs in a large proportion of cases and may be of rapid development.

**Physical Signs.**—The signs are those of fluid in the chest and are consequently similar to those described under the heading of sero-fibrinous pleurisy. Empyema, however, is more common in young children and more often associated with lung consolidation of an inflammatory nature, and, as a consequence, those signs—bronchial breathing and sometimes bronchophony—which may simulate a simple pneumonia are commonly present, whereas, in the serous pleurisy of older children the signs are often similar to those of effusion in the adult. Moreover, loculation of the effusion more often occurs with empyema, so that the signs may be found only over a limited area of the chest, generally behind, giving a still closer resemblance to pneumonic consolidation. In cases where the fluid is free it tends to gravitate to the lowest parts of the pleura, and, not uncommonly, a whole side is found dull to percussion, the signs at the upper part being due to solid lung, which gives intense tubular breath sounds and bronchophony, and the base being occupied by an empyema, the breath sounds becoming feebler as one descends the chest wall, though retaining their bronchial character wherever audible. In effusions of no great size the signs are often widely distributed, a thin layer of pus stretching up between the pleural surfaces.

When only a thick layer of fibrin is present, a condition which lies on the borderland between empyema and serofibrinous pleurisy, the signs may be but slight, but generally there are dulness, feebleness of breath sounds, and sometimes a "glutinous" pleural friction sound. They depend in most part on the condition of the lung beneath, which may be that of consolidation, or of partial collapse.

Apical empyema is sometimes met with, but is rare, and is more often diagnosed than found.

Among 81 cases of empyema collected by Mr. P. S. Blaker at the East London Children's Hospital, 42 occurred at the left base and 30 at the right base; 9 were double. From another source more cases were recorded on the right side than on the left, from which it appears probable that the disease shows no obvious partiality for one side above the other. It will be remembered that in croupous pneumonia which so often precedes this disease the right and left lungs are affected in a nearly equal number of cases.

It are forms of empyema are interlobar and diaphragmatic collections. Thick layers of lymph are, however, not uncommonly found in these situations with empyemata of wider extent.

**Complications.**—Uncomplicated empyema, unless in very young infants, is seldom found in the autopsy-room. The disease generally kills through its complications and these are all of a serious nature. In fatal cases caused by the pneumococcus it often happens that many

lesions are associated, especially multiple infection of serous cavities (pericardium, pleura, and peritoneum), and often in such cases no one affection can be considered primary, the disease being probably a blood infection from the start, a pneumococcic pyemia.

*Bronchopneumonia* is perhaps the commonest complication and often leads to death. Doubtless, cases of empyema are sometimes secondary to bronchopneumonia, but I am inclined to believe, myself, that more often it is the bronchopneumonia which is subsequent. It generally affects the partially collapsed lung beneath the empyema and often leads to extensive consolidation. The opposite lung also is affected in many cases.

*Purulent pericarditis* is found in a large number of fatal cases, both it and the empyema being often secondary to pneumonia. It has been already described under the heading of lobar pneumonia (p. 640).

*Purulent meningitis* is an occasional and fatal complication. It is often present in association with other infective lesions, especially suppurative pericarditis. Layers of greenish fibrin are found covering the vertex and extending in some cases over the whole surface of the brain. The symptoms are often obscure in young children, and if pneumonia is also present they may be ascribed to the cerebral symptoms of this disease. Convulsions are the most marked symptom, but if the base is also involved paresis of cranial nerves may arise. Occasionally an unusually slow pulse rouses suspicion when no other symptoms are present.

*Purulent Peritonitis.*—A suppurative peritonitis is found in some fatal cases of empyema in children, most often so in cases of widespread infection to which the name of pyemia might be given. The symptoms pointing to the abdomen may be so slight as to be overlooked among those implicating other organs, but, when sought for, abdominal distention and the ordinary signs of peritonitis may generally be found.

*Cellulitis.*—Not uncommonly there is some inflammation of the cellular tissue of the mediastinum. This may reach such a grade that the heart and pericardium are surrounded by a layer of pus, which may spread back over the vertebrae and round the chest wall between the parietal pleura and the ribs. In some cases the mediastinum is unaffected, but there is a layer of pus spread out over a wide area of the chest wall between ribs and pleura, generally stretching forward from the bodies of the vertebrae. I have observed such a cellulitis of the chest wall without empyema as a complication of bronchopneumonia.

*Tuberculosis.*—Empyema sometimes occurs as a complication of chronic pulmonary tuberculosis in children, and, in rare instances, has been associated with the presence of a pneumothorax. Occasionally the empyema is primary, and the child is attacked by a subsequent acute general tuberculosis which leads to death.

*Termination.*—If an empyema is overlooked and remains untreated as a rule, the child wastes and dies, but three possibilities are open apart from this. The empyema may dry up, leading to fibrosis of the underlying lung and bronchiectasis, with retraction of the chest wall.

It may ulcerate into a bronchus and be coughed up, though this happens more rarely in children than in adults; a cure may be thus effected, but more commonly the discharge continues until the condition is recognized and treated. When such an occurrence is suspected, it must be borne in mind how closely the condition may be simulated by a fibrotic lung with bronchiectatic cavities, from which large quantities of pus, indistinguishable from the pus of empyema, may be expectorated.

In a few cases the abscess may open through the chest wall, though this appears to be uncommon in childhood; it may open at any point, but generally does so in front; on the other hand, the pus has been known to perforate the diaphragm and appear as a psoas, iliac, or gluteal abscess.

**Sequelæ.**—In cases where complete obliteration of the empyema cavity has failed to occur, owing to deficient expansion of the lung and inability of the chest wall by its contraction to meet this deficiency, a discharging sinus remains, and will not heal until operative measures, such as Estlander's procedure, have closed the gap. Occasionally, again, a sinus is caused by too long retention of drainage tubes. Such a discharging sinus, if left untreated, will lead to ill-health, and may even cause amyloid changes in the viscera.

In cases where an empyema has been neglected so that the lung remains collapsed during a long period, and also where thick layers of lymph have remained and become organized, *pulmonary fibrosis* is liable to occur. The side of the chest becomes retracted, and bronchiectasis is set up in the lower lobe of the lung.

**Diagnosis.**—The first point in the diagnosis is generally that between solid lung and pleural effusion. This has been left until the present rather than discussed under the heading of serofibrinous pleurisy, because empyema is more liable to be mistaken for lung consolidation (and *vice versa*) than is serous effusion, owing to the earlier age of the child, the association of the disease with pneumonia, and the greater prevalence of loculation of pus than of serum in the pleural cavity.

The diagnosis of a serous effusion from pneumonia seldom presents any difficulty. The position of the fluid round the base of the chest presents an unmistakable picture, as does also the marked displacement of neighboring organs when a large effusion is present. It is a loculated empyema that may be so readily mistaken for pneumonia, and this may occur in any situation save that it is rare at the apex. If the signs are limited to the apex a diagnosis of solid lung is more likely to be correct, even though the signs incline to simulate those of fluid. When at the base, the signs are often deceptive and many points need consideration.

**Inspection** helps little; the side may move well with fluid or, on the other hand, its motility may be impaired with solid lung. **Palpation** may give a valuable clue, since a fluid thrill is sometimes obtainable. This is tested by placing a hand over the base behind while an assistant percusses sharply in front, the sensation obtained being compared on the two sides. A vocal fremitus is not obtainable in young children.



On *percussion* the dulness of fluid is more "wooden" than that of solid lung, and the sense of resistance to the finger greater. This is the most important sign, though in some cases massive consolidation will impart a sensation of considerable resistance. The shape of the dull area helps in certain cases, especially in its relation to the lobar divisions of the lung.

*Auscultation.*—We have seen that in children loud, bronchial breath sounds may be heard over an empyema; more often they are somewhat distant, though bronchial in quality, and they often become fainter as the base of the chest is approached. Even when loculated, fluid tends to gravitate downward, and the increase in dulness and tactile resistance, and the loss of breath sounds at the lowest point may be signs of great value in differential diagnosis; in pneumonia, on the other hand, the signs of most advanced consolidation are more often found above the lowest point. Distant breath sounds at the base are, therefore, of importance, especially if these become progressively louder as the chest wall is ascended, but it must not be forgotten that the breath sounds may be suppressed for a while over solid lung also. The vocal resonance over an empyema is increased where bronchial breath sounds are loud, and, where these are distant, which is generally at the base, it is diminished or lost; its value in this respect consequently follows closely that of the breath sounds. There is another point, however, which may lend importance to the vocal resonance—a nasal quality is often audible over effusions and is a valuable distinction when present. The simulation of pneumonia by effusions in children is due in no small part to the presence of a consolidated or firm, collapsed lung lying behind the fluid layer.

Among general symptoms the anemic and straw-tinted complexion of the child with empyema, the more moderate rapidity of respiration, the lower temperature level, and often a "glazing" of the finger-nails preceding clubbing, may lead to a correct diagnosis where the signs in the lungs are of doubtful significance.

An examination of the blood is often of definite value; a high leukocyte count is more likely to indicate a purulent than a serous effusion, and a sudden increase in leukocytes during the course of a pneumonia points to an empyema. Such blood examinations should never be neglected in these cases.

When all these things have been taken into consideration there still remains one test which should never be omitted in case of doubt, namely, exploratory puncture of the chest wall. In some cases this is the only means of deciding the diagnosis, and even here failure is not infrequent owing to blocking of the needle with fibrin and the difficulty of extracting fluid under negative pressure. A large-sized needle should be used and only a positive result accepted as conclusive.

*Pus or Serum.* Having decided upon the presence of fluid in the chest some attempt may be made to decide whether this is clear fluid or pus; in some cases the distinction is impossible, in others there may be little doubt. Thus, in older children the probability is in favor of serofibrinous pleurisy, and if the history is short, the color good, no



suspicion of finger clubbing present, and if the fluid is not loculated, a diagnosis of this disease will be confidently made.

On the other hand, if the child is below the age of three years, especially if it is an infant, and shows marked anemia with a yellow-tinted skin, if the history is long, and there is clubbing or great shininess of the finger-ends a diagnosis of empyema will be made with equal confidence, and this especially if the effusion is localized.

Both the general symptoms and the physical signs must be considered in making a diagnosis between pus and serum. The *age* is of importance. Empyema is more common in infancy (after six months of age) and becomes much less so as years advance; among 81 cases at the East London Hospital for Children, nearly 35 per cent. occurred during the second year, falling to 10 per cent. in the third year. Serofibrinous pleurisy, on the other hand, is rare in infancy and becomes more common when later childhood is reached. The *duration* of the effusion is of some help in determining its nature; in most cases a serous effusion begins to be absorbed within ten days or two weeks of the onset; empyemata generally remain in *statu quo* until treatment is adopted. When treatment is delayed anemia is rapidly developed, the muscles become flabby and waste, and the face, and often the body, assumes a yellowish tint. Finger clubbing, when found, is an important point in favor of purulent effusion; its commencement is occasionally appreciable within a week of the onset, the first change being generally a peculiar shininess of the skin over the terminal phalanges.

The physical signs in the chest usually give less indication of the nature of the effusion than do the general symptoms. A loculated effusion is always more likely to be purulent, except perhaps in the region of the right middle lobe in front, where serous effusion sometimes starts in children and remains for a while localized. Tenderness on percussion is more often noted in empyema than in serous effusions, and edema of the chest wall is a valuable sign of pus, but is very seldom found. Pus is said to give a denser shadow to the x-rays than does clear fluid. As a rule, the diagnosis between pus and serum at the present day is rapidly put to the final test of exploration without much trouble being expended on more subtle points of diagnosis. Sometimes the exploring needle draws off a fluid of doubtful nature; it may be just opalescent, and a clot may form which is less transparent than usual. Microscopically, pus cells are found in moderate numbers and perhaps on culture a few colonies of pneumococcus develop. Such cases generally progress to pus, but in a few cases they undoubtedly recover without going farther. Cases with turbid fluid are cases of empyema and should be treated as such; in many of the most acute and fatal cases the fluid is only thin and turbid. There is no doubt at all that a clear serous effusion may become purulent, but such a change is of very rare occurrence; when it occurs the pneumococcus was generally present from the first.

*From Fibrosis of the Lung.*—The diagnosis is considered under the heading of that disease (p. 678).

**Prognosis.**—This depends on the age and general condition of the patient and the promptitude with which treatment has been carried out, but also largely upon the origin of the empyema.

The disease is very fatal in the first year of life. Among 81 cases of empyema treated at the East London Hospital for Children 11 were in infants below one year of age, and of these only 1 recovered. The condition is, however, not necessarily hopeless in the youngest children, since a case has lately recovered in the same hospital at the age of four months; such an occurrence, however, is exceedingly rare. After the first year is past the chances are much better. The total mortality among these 81 cases quoted was 38 per cent.; the mortality for cases over one year of age was only 28.5 per cent.

The general condition of the child is necessarily of great importance in estimating the chances of recovery, and this weighs mostly in those cases where intense anemia with wasting and general asthenia are due to the presence of an untreated empyema. Such a case means, of necessity, a prolonged convalescence owing to imperfect expansion of the lung.

With regard to the influence of the bacteriological result on the prognosis, it may be said at once that the pneumococcal cases taken as a whole are by far the most favorable. Of my own cases, mentioned under the tabular report, p. 654, all those due to the staphylococcus died, 2 of them showing a definite septic source from which the infection had sprung. Of the 3 streptococcal cases, 1 recovered, 1 died, and 1 was removed from the hospital in *statu quo*. Of the 3 cases of mixed pneumococcus and streptococcus infection 1 was cured and 2 died, and among the 2 cases in which the pneumococcus, staphylococcus, and a doubtful bacillus were found, 1 recovered and 1 died. Thus, among these 11 cases, in which other organisms besides the pneumococcus were present, 7 or probably 8 died, and only 3 recovered. It is interesting that 2 of these cured cases were streptococcal, and I find another writer mentions the fact that 4 cases of streptococcal origin among 40 cases of empyema collected by himself all ran a mild course to recovery. Thus it appears that the streptococcus as a cause of empyema in children is not such a virulent organism as we should, on other grounds, have expected.

The pneumococcus cases in my series showed a much lower mortality, only 30 per cent. among the 66 cases. This includes children of all ages, and it is obvious, from what has been said above, that an uncomplicated single empyema in a child above the age of one year is, on the whole, of good prognosis. Among pneumococcal cases, those in which the lung consolidation and the effusion begin simultaneously are more often fatal than those in which the empyema occurs as a later complication of pneumonia. Cases complicated with infection of other serotypes or membranes are almost of necessity fatal; such cases are often of the nature of a pneumococcal pyemia and in many the infection is probably general from the beginning.

The position of the lesion matters little; the size is of less importance.

than might be expected, large empyemata generally doing well, perhaps because they are more promptly recognized. Double empyema is distinctly unfavorable.

Some writers affirm that indications of value may be gathered from the microscopic characters of the pus. A small number of cocci in the films is regarded as favorable, a large number unfavorable; imperfect staining of the organisms, moreover, is judged of good omen, as is also phagocytosis, when this is seen.

**Treatment.**—The treatment of empyema is the treatment of an abscess, though its peculiar relations to the thoracic organs renders it an abscess of a rather special kind. *Aspiration* is not generally a success, and should only be used as a palliative measure, since the cavity nearly always refills. Where the effusion is large and causing much respiratory difficulty, and especially if for any reason delay in operating must occur, it is well to draw off a large quantity before the operation is performed. Where more radical treatment is refused aspiration must be tried as a curative measure. In double empyemata an operation should be performed at first on one side only, the other pleural cavity being emptied or partially emptied by aspiration. This may be repeated if necessary; in any case the operation on the second side should be left as long as the conditions allow, if possible until the first is nearly healed.

The indication in empyema being for immediate free drainage as soon as the condition is recognized, the only question is the best method of doing this, the choice lying between *simple incision* between the ribs and *resection* of a portion of rib to allow more space for drainage. In young infants, under a year or even under eighteen months of age, in whom operative measures are not well borne, simple incision is to be preferred, and, as a rule, the drainage afforded is amply sufficient. The same applies to older children, where the condition is too serious to admit of an anesthetic being given; if drainage prove insufficient a portion of rib can be removed subsequently. The farther forward the incision is made, the more space there is between the ribs; the further back, the better the drainage in the supine position. A point must be chosen where these advantages meet. An incision one and a half inches long in the ninth space at the posterior or midaxillary line is generally convenient from both points of view.

In older children the removal of a portion of rib is usually necessary to establish free drainage. The eighth or ninth rib in the posterior axillary line is usually chosen, the rib is cut down upon, the periosteum incised and separated, about one inch of rib removed with bone forceps, and the pleural cavity opened above the incised periosteum. The liquid pus and as much thick fibrin as can be reached with the finger should be removed. The operation should be performed as soon as pus has been found with the exploring syringe, the only exception perhaps being certain cases where the symptoms are severe owing to extensive lung consolidation at the onset, and where the empyema is adding little or nothing to the gravity of the condition. Under such circumstances



delay, or aspiration as a temporary measure, may be wisest. An apical empyema can generally be reached both for exploration and for drainage, from the apex of the axilla.

**After-treatment.**—Having opened the empyema a large-sized rubber tube is introduced, or, with simple incision, two smaller tubes side by side as large as will pass between the ribs. The wound is dressed with aseptic dressings and layers of wool to absorb the discharge, which may be considerable at first. The drainage tube should be removed daily, boiled, shortened, and reinserted. It is a common mistake to retain it too long, causing in many cases the formation of a troublesome sinus. It may often be dispensed with at the end of a week, a gauze drain being left in for a few days and a sinus forceps passed in when the wound is dressed to let out any pus that may be retained. If drainage seems inefficient without it, the tube may be reinserted for a few days. After first leaving out the tube, it is a good plan to employ a wet dressing for a few days to assist the exit of pus and prevent too early closing.

The surrounding skin should be kept clean and dry, an occasional washing over with ether being a good plan. As a rule, there is no need for irrigating an empyema cavity; if, however, the pus is foul the cavity may be safely washed out with a solution of tincture of iodine, 4 c.c. to 475 c.c. (1 dr. to the pint), but no force must be used. The coughing caused by injection is very efficient in removing thick collections of pus.

The child should be got up as early as possible so as to aid the expansion of the lungs by movement, and in older children this object may be furthered by an ingenious arrangement of bottles in which colored water is blown from one into the other; in private a trumpet or some other form of wind instrument may be used to incite the patient to expiratory efforts, for it is by expiratory pressure that the inflation of the collapsed lung is brought about.

Many cases heal in three weeks, but the majority take longer, two months being a not uncommon time limit. When the cavity does not close this is due to inability of the lung to expand, and for this some definite cause can generally be found. In some the condition has remained too long before an operation was performed, and the lung is permanently bound down by adhesions, by fibrous change, or by layers of organized fibrin enclosing its surface; in others there is chronic tuberculosis, which will not permit the full expansion of the lung.

When the lung will not expand the chest wall must be made to give in, and to aid this various operations have been devised, notably Estlander's, in which portions of several ribs are removed; or Shedden's, in which parts of the pleura and intercostal muscles are also cut away to contract the side still further. In some cases also the thick layers of organized fibrin have been stripped from the surface of the parietal pleura, thus allowing the enclosed lung to expand freely.



**PNEUMOTHORAX.**

This is a rare disease in childhood, since phthisis, which forms the common cause in the adult, is of uncommon occurrence in children. The majority of cases in children appear, nevertheless, to be tuberculous in origin, other causes being pulmonary gangrene, infarction, emphysema, sometimes in association with whooping-cough, foreign bodies in the bronchi, fractured ribs, empyema, and bronchiectasis. Cnopf describes three cases occurring during the course of diphtheria with laryngeal stenosis, and in some of these emphysema of the mediastinum and subcutaneous tissues also occurred. A case due to tearing of the lung by an adhesion during coughing has also been recorded.

**Symptomatology.**—In such cases as occur suddenly there are shock, great prostration, dyspnea, chest pain, and weak, rapid pulse. When of gradual onset the dyspnea and pain will be less severe. Vocal fremitus is absent over the pneumothorax, but may be normal or increased over the compressed lung. There is a tympanitic note over the air cavity, but if the pleura is very tense this will be less marked, or even simulate dulness. Voice sounds are distant; over the compressed region there is an amphoric or tubular character to the respiration. Coins that are placed over the tympanic area cause a distinct metallic sound when tapped together.

The heart and liver may be displaced.

When there is fluid as well as air in the pleural sac the metallic tinkling and slushing must be differentiated from fluid in the stomach.

**Treatment.**—The treatment is essentially that of pleurisy, with effusion. Strapping and compression of the chest may give some relief.

## CHAPTER XXVII.

### ABSCESS OF THE LUNG—GANGRENE OF THE LUNG—BRONCHIECTASIS AND PULMONARY FIBROSIS—FOREIGN BODIES IN THE AIR TUBES.

#### ABSCESS OF THE LUNG.

LUNG Abscesses are not uncommonly seen on the autopsy table as small multiple foci in the midst of pneumonic consolidation, but as a clinical condition the disease is rare. It occurs in young and weakly children, usually as the outcome of a croupous pneumonia, but sometimes of a bronchopneumonia.

**Symptomatology.** The symptoms develop themselves gradually out of those of the primary disease. Thus, an attack of pneumonia pursues its ordinary course and the crisis occurs as usual, but the temperature rises again and becomes hectic. The bronchial breathing, perhaps, disappears over the pneumonic area, but the dullness remains and becomes more marked, so that a loculated empyema is usually suspected. The child loses weight and color, sweats profusely in most cases, and appears seriously ill. The temperature generally shows wide fluctuation, rising high at night and falling to normal or subnormal in the morning, and, on examining the blood, a leukocytosis is discovered, perhaps as high as 40,000 or 50,000 per cubic millimetre.

**Physical Signs.**—The signs closely simulate those of loculated empyema, namely, dullness, with marked increase of resistance, and feeble breath sounds, often bronchial in quality, but, in addition, coarse râles and friction sounds are generally audible. The abscess develops in the site of the original pneumonia, and hence may be found either in the upper or in the lower lobe of the lung.

**Diagnosis.**—In the early stages the condition appears to be nothing more than *unresolved pneumonia*, but soon the continued fever, wasting, and anemia, and the generally unsatisfactory progress point to something further, the higher leukocytosis being the most important point in the differential diagnosis.

When pus has collected an *empyema* is nearly always diagnosed and an operation for that disease may reveal the true condition, but an abscess of the lung may be drained under the belief that empyema only is being dealt with. The diagnosis from empyema is often very difficult, since the marked leukocytosis is common to both. Pleural friction occurring over the dull area is no guide, since in empyema a friction rub is often audible, being probably produced over contiguous solid lung. The absence of displacement of the surrounding viscera with pulmonary abscess may be of value in some

cases, but its occurrence is not constant. I have seen the heart pushed over by an abscess of the left lung so that its apex was close to the middle line of the chest.

**Treatment.**—The disease runs a long course, often of many months' duration, and, if untreated, generally ends in death. Expectoration of the pus does not commonly occur. The treatment is operative, as for abscess elsewhere. Pus has generally been discovered with the exploring syringe, often with difficulty, and a rib resection is undertaken under the impression that an empyema is present. A healthy pleura being discovered, if the surfaces are non-adherent, which is unlikely, the opening must be packed with gauze, or the lung stitched to the opening so that adhesions shall form and allow of further procedure after a few days' interval. Where adhesions already exist the further operation may be proceeded with at once. This consists in again exploring for pus, and, when it is found, opening the abscess by boring through the lung with a Pacquelin cautery at dull-red heat or, in absence of this, with a dressing forceps. The pus, seldom more than one or two ounces in quantity, is allowed to escape, a drainage tube inserted, and similar after-treatment pursued as in a case of empyema. Syringing the cavity should be avoided if possible, as this proceeding may give rise to considerable shock.

#### GANGRENE OF THE LUNG.

This is a rare condition and, when it occurs, it often remains undiagnosed during life.

**Etiology.**—The disease arises as a result of many varied processes. A number of cases originate in bronchopneumonia, especially when this complicates typhoid fever, measles, or others of the infective fevers, or when an "aspiration pneumonia" occurs, generally as the result of a tracheotomy. Croupous pneumonia originates some cases, and many are the result of a septic embolism, as in cases of lateral sinus thrombosis following mastoid disease. Bronchiectasis is a not uncommon cause, especially when this is due to the presence of a foreign body in the bronchus, a septic bronchopneumonia usually preceding the gangrenous process. The condition is sometimes secondary to cancrum oris.

**Pathology.**—The gangrenous areas are very commonly multiple. When complicating pneumonia they may be large in size, two or more inches in circumference in many cases, and are surrounded by consolidated tissue showing the characters of bronchopneumonia or croupous pneumonia; when due to septic embolism they are generally scattered and isolated, perhaps the size of peas or cherries. They are dark brown, green, or black in color, and consist of soft, shaggy material, of putrid odor, sometimes leaving a cavity in the centre containing blood clot or liquefied debris. The gangrenous areas are generally found near the surface of the lung, and there may be some pleurisy over them, or an empyema may have arisen. Softening thrombi are often present in the venous channels draining the affected part.

**Symptomatology.**—Symptoms are generally obscure and may give no clue to the nature of the condition present. If the condition arises in pneumonia, the symptoms of that disease are prolonged, the temperature fluctuates, the cough becomes paroxysmal, there may be chills and sweating, and the child wastes and becomes extremely ill. None of these symptoms is diagnostic of the condition, and these alone may be present; but in other cases a gangrenous odor of the breath arises, and the characteristic sputum, perhaps, appears—green, or dark brown in color, blood-stained, with a putrid odor, and showing fragments of lung tissue to microscopic examination. Not uncommonly hemoptysis occurs and may prove fatal, as in a case occurring at the East London Children's Hospital, where over a pint of blood was expectorated immediately before death. The fetid breath or sputum and the hemoptysis, when these occur, should enable a diagnosis to be made, in spite of the fact that both may be found in certain cases of bronchiectasis with pulmonary fibrosis. It is important to remember that among cases of gangrene in children a large proportion show no fetid odor of the breath.

**Physical Signs.**—The physical signs are often those of pneumonia, but they may be those of bronchitis only in cases where septic emboli have given rise to multiple gangrenous areas too small to produce signs of consolidation. When an abscess has formed within the gangrenous area and the contents are expectorated, signs of cavity, amphoric breathing, and pectoriloquy may sometimes be present. In some cases the abscess breaks into the pleural cavity, giving rise to empyema often accompanied by pneumothorax, when the signs of these diseases will be present to examination. In such cases, if the gangrenous area is single, the slough may escape with the pus when operation is performed and a cure result.

**Treatment.**—This consists in operative measures as soon as the nature of the condition is recognized. The results of this treatment are very encouraging; Seitz reports 61 per cent. of recoveries among such cases and remarks that those in which the gangrene follows croupous pneumonia offer the most favorable prognosis. The steps of the operation required are similar to those mentioned under abscess of the lung. At the same time the child's strength must be supported by tonic remedies and a liberal diet, accompanied by alcoholic stimulants, and the fetid odor controlled by the use of inhalations of eucalyptus, creosote, and others of the volatile antiseptics.

#### BRONCHIECTASIS AND PULMONARY FIBROSIS.

Bronchiectasis is so intimately associated with Fibrosis of the Lung in children that it is convenient to consider it in connection with that disease rather than with bronchitis, where its more natural place would seem to be. Fibrosis is always accompanied by bronchiectasis, and bronchiectasis often gives rise to fibrosis, its presence in nearly all cases being accompanied by some induration of the surrounding lung tissue;



at the same time when this fibrosis is small in amount the bronchiectasis stands by itself as a clinical entity, and as such demands separate description.

**Bronchiectasis.**—Bronchiectasis may be either *temporary* or *permanent* in character. Some amount of dilatation occurs during the course of a prolonged bronchopneumonia or bronchitis and may largely or entirely disappear. The amount of recovery depends upon the duration of the attack and the depth of the inflammatory process; when these are considerable some permanent enlargement of the tubes is left.

The dilatation is either cylindrical, fusiform, or sacculated, the last being generally secondary to fibrosis of the lung.

**Etiology.**—The disease usually dates from an attack of bronchopneumonia or bronchitis, very commonly in association with whooping-cough or measles, and generally affects both lungs, though it is nearly always most marked on one side. In rare cases the condition is said to be congenital.

The pathogeny of the disease has been the subject of much controversy, but two causes seem to be at work in most cases. The most important of these is a *softening of the bronchial wall* due to inflammatory changes. This is well illustrated by certain rare cases where dilatation of the smallest tubes throughout the lung—acute bronchiolectasis—has occurred. In these the microscope reveals a peribronchitis associated in some cases, but not in all, with bronchopneumonia. The other cause is *strain* from increased pressure due to cough, especially in the violent paroxysmal attacks associated with pertussis.

**Symptomatology.** After a prolonged attack of bronchopneumonia or bronchitis, especially when associated with pertussis, the cough persists and the child is found to bring up a considerable quantity of sputum daily. It may be expectorated with cough, but often large quantities are expelled by vomiting, especially in young children. Attacks of coughing generally occur at long intervals and are violent and paroxysmal. The sputum is usually green pus and sometimes has a stale odor; it may, in rare cases, become offensive, but this more often occurs where the condition is associated with fibrosis of the lung. The cough may persist all the year round, the amount of expectoration being greater at times, especially in the winter; in other cases the secretion stops for a while in the summer months or is brought up only once daily, generally on rising in the morning. The general health may be little affected, and, though the children often waste during the winter months, they generally grow fat under treatment, or in the favorable periods of the year.

**Physical Signs.**—The signs, when any are present, are those of bronchitis, with, in addition, the element added by the bronchial dilatation. In most of the cases without fibrosis no definite signs of cavity are found, but the râles are large, moist and bubbling, and at certain parts of the chest, especially at the bases behind and in the axillæ, may have a resonant quality accompanied by harsh or even bronchial breath sounds. At times the râles have a dry, rustling quality, not unlike fine

friction. The vocal resonance is often unaltered, but there may be bronchophony at some favorable spot. In the warm months of the year the mucous membrane of the dilated tubes secretes but little, and the signs nearly or quite disappear, a few dry rhonchi perhaps being heard over the chest.

**Diagnosis.**—The diagnosis from bronchitis is generally determined by the amount of expectoration and by the character of the râles—their large, bubbling nature, and the resonant quality lent to them by the cavity in which they are formed. In bronchiectasis, too, the signs are more localized and generally more marked on or even confined to one side of the chest.

In any case of marked bronchiectasis, if one-sided, whether with or without definite fibrosis, the possibility of foreign body as a cause must not be overlooked.

**Prognosis.**—The *complications* and *prognosis* are largely those to be dealt with under the heading of pulmonary fibrosis, save that in the absence of fibrosis the dilatation of the tubes is seldom so marked and their contents are more easily expelled. As a result of this the expectoration more rarely becomes putrid, and thereby a source of ill-health and of great danger is avoided.

**Treatment.**—The general health must be attended to on similar lines to those about to be described under pulmonary fibrosis. For the local conditions the emptying of the cavities and the relief of the fetor, at present, are the most important considerations. For the former the effect of posture may be taken into account with great advantage. By hanging the head and chest over the edge of a bed the cavities empty by gravity into the more healthy tubes above, violent cough is induced and great quantities of phlegm may thereby be expectorated. This practice should be employed at regular intervals to drain the passages—two or three times daily being sufficient in many cases. In addition to this an occasional emetic is of service for the same purpose, and at times when the sputum is very free a stimulant expectorant may be given, such as the following, to a child of three or four years, at four hourly intervals:

<b>R</b> —Ammonii carbonatis . . . . .	0.05 gm.	(gr. 3)
Tincture scillæ . . . . .	0.12 c.c.	(m. 4)
Syrup. toluant . . . . .	1.50 c.c.	(m. 50)
Infus. senegæ . . . . .	4.00 c.c.	(15)
Aq. . . . .	q. s. ad 8.00 c.c.	(3 <i>℥</i> 1 <i>℥</i> M)

If the expectoration become foul, attempts must be made to purify it by the inhalation of volatile antiseptics, such as creosote, thymol, eucalyptol, etc. They may be inhaled undiluted from the surface of hot water, or from a respirator in the strength of 4.0 to 8.0 c.c. (1 or 2 dr.) to 30 c.c. (an ounce) of spirits of chloroform, or carbolic acid may be used as a spray in 2 to 4 per cent. solution by means of an atomizer. The air of the living-rooms may also be kept sweet with a spray of any of these volatile oils dissolved in rectified spirit (1 in 6).

More efficient is the method for creosote inhalation introduced

Dr. Arnold Chaplin. The creosote is vaporized in a small room and the patient endures the vapor for increasing periods daily, from one-quarter hour at the beginning up to one hour at a sitting. The nostrils are plugged with cotton-wool, and the eyes protected with watch glasses framed in sticking plaster. The effect of the strong vapor is to cause effective coughing so that large quantities of foul sputum are expectorated, and, in addition, disinfection of the emptied cavities takes place.

External drainage of bronchiectatic cavities has been performed, occasionally with success, but more often with failure. Its employment

FIG. 142



Fibrosis of lung in a girl of four and a half years; shaded area represents the contracted left lung; the hypertrophied right lung crosses the middle line by one inch; the position of the heart's apex is indicated by a cross.

is mainly suitable to those rare cases where a single large cavity forms the bulk of the trouble; as a rule, the dilatation is widespread.

**Pulmonary Fibrosis.**—Fibrosis of the lung of slight grade is by no means uncommon in children, but is often overlooked. Many of these slighter cases are the result of a former whooping-cough and belong, perhaps, rather to bronchiectasis than to fibrosis, since the dilatation of the bronchi is the important lesion. The more marked examples of pulmonary fibrosis generally own a different etiology, and present a



a very typical clinical picture which is not uncommonly mistaken for pulmonary tuberculosis.

Fibrosis as a process of repair is a common accompaniment of many pulmonary lesions; it is only when it occurs on a wide scale that its presence is recognizable during life, and, though it does not constitute a disease in itself, the train of symptoms and signs brought about by its presence form a very definite clinical picture, and make it desirable to group many cases of different etiology under this one heading.

**Etiology.**—The following table prepared from 32 of my cases, in which a clear history could be obtained, shows roughly the common antecedents of pulmonary fibrosis.

Bronchopneumonia, alone . . . . .	6
" with whooping cough . . . . .	5
" with measles . . . . .	3
	— 14
Lobar pneumonia . . . . .	4
Bronchitis, alone . . . . .	5
" with whooping-cough . . . . .	3
" with diphtheria . . . . .	2
" with measles and whooping-cough . . . . .	2
	— 12
Congenital atelectasis . . . . .	2
	— 32
Influence of the infective fevers among these cases:	
Whooping-cough, with bronchopneumonia . . . . .	5
" with bronchitis . . . . .	3
" with measles . . . . .	2
	— 10
Measles, with bronchopneumonia . . . . .	3
" with whooping-cough . . . . .	2
	— 5
Diphtheria . . . . .	2
	— 17

It is clear that the majority of cases originate in bronchopneumonia and bronchitis, and that especially when these are complications of whooping-cough or measles. When bronchopneumonia is the starting point the attack drags on to great length, the signs never quite clear, and when health returns a certain amount of fibroid change is left in the lung. Often, after an interval of good health, another attack of pneumonia ensues and the lung is still further crippled. These successive attacks may arise at intervals. In the early stages, could we inspect the lung, we should find a dilated bronchial tree with thickened walls, such as we find after any protracted bronchopneumonia, and, as the attacks proceed, inflammation leading to fibrosis spreads from the tubes into the lung, the process being probably aided by fibrosing areas of unresolved consolidation and of collapse. When the fibrosis has become considerable saccular cavitation of the smaller bronchi appears. Where lobar pneumonia originates the condition, one lung only is affected, generally part of a lung, and the process may be limited by quite a sharp line of demarcation. Lobar pneumonia is doubtless responsible for most of the apical cases.

In cases arising in bronchitis I have observed that the right middle lobe is very commonly affected, and, seeing the comparative frequency



with which collapse occurs in this part of the lung in bronchitis, I am disposed to attribute the fibrosis in many of these cases to such collapse.

Next come cases which originate in earliest infancy and are probably due to *congenital atelectasis* of a portion of lung. The symptoms may not appear till some six months later. Lastly, an untreated *pleurisy* occasionally leads to fibrosis of the lung, but less often, I think, than is generally held. When this occurs it is in those cases where thick layers of fibrin have been left to become organized in the pleural cavity.

Pulmonary fibrosis is found at all ages throughout childhood, but its origin is most commonly traced to the early years of life. The following table shows the age incidence among 38 cases coming under my own observation:

Below age of 3 years . . . . .	2 cases.
3 to 5 " . . . . .	5 "
5 to 10 " . . . . .	15 "
10 to 15 " . . . . .	13 "
15 to 22 " . . . . .	3 "
	<hr/> 38

Of the 2 cases occurring below the age of three years, in one, aged one year and ten months, the disease was verified by autopsy.

**Pathology.**—On opening the chest the lung, if affected as a whole, is found lying far back in the chest; the mediastinum and heart are drawn over to fill the vacant space, and with them the opposite lung, which is voluminous. When only part of a lung is affected these changes are, of course, much less marked. The lung or its affected part is, as a rule, closely adherent to the chest wall, though the pleura may be a little thickened except in rare cases where the process is pleural in origin. When the process is advanced the lung tissue is firm, tough, and elastic to cut; dark, slate-color, or pinkish gray on section, and totally airless. Through it run the dilated larger tubes; the smaller tubes form saccular cavities throughout its substance and these often contain foul pus. Their walls are red, smooth, and glistening. The fibrotic area may be sharply circumscribed, or the remainder of the lung may show a less advanced change, namely, some increase of fibrous tissue and moderate dilatation of the bronchi, their walls being thickened and showing as white lines through the section. In cases which are primarily and perhaps principally bronchiectatic, the tubes are thickened and much dilated in the lower lobes and root of the lungs, but some fibrous change is generally to be observed around them.

The lymph nodes connected with the lung are enlarged and generally show on section much pigmentation, but little or no fibrosis; they are often quite soft and pulpy.

**Histology.**—The process by which a pneumonic area becomes converted into fibrous tissue is double. There is an invasion of the alveolar walls by connective-tissue cells, and also a replacement of the alveolar exudation by spindle-shaped cells with obliteration of the lumen. The same process occurs where bronchiectasis is the starting point, fibrous tissue replacing the inflammatory area around the dilated tube and

spreading with each exacerbation farther into the lung. When the condition is complete, the lung presents to microscopic examination strands of fibrous tissue, young or old, but mostly cellular and nucleated, winding in various directions, and enclosing numerous blood spaces. Dilated bronchial tubes are seen here and there, some of them showing a round-celled infiltration of their walls. Little or no healthy lung tissue may be visible.

**Symptomatology.**—The onset of the symptoms varies with the cause. When this is bronchitis the process is of slow development and its stages ill-defined, the lung changes increasing with each acute catarrh. In cases beginning with bronchopneumonia there is usually a frank attack of this disease, and occasionally it appears to resolve normally, but generally resolution is delayed and some signs, a little impairment, and some râles are left. A few months later the child may again be seized with pneumonia, which settles in the same parts of the lung as were involved during the first attack. The illness is protracted over two or more months; the child remains somewhat weakly or regains his normal health, but is left with permanent lung signs. Further acute attacks may supervene and in young children the conditions tend to get worse and worse, the child sometimes dying in one of the acute attacks or from some complication to be described later. In other cases, especially when later childhood is reached, he may regain his vitality and grow up with a lung or part of a lung contracted, but with fair general health. When lobar pneumonia is the starting point of the disease a somewhat similar picture of repeated attacks may be produced, or the fibrosis may originate in a single unresolved consolidation.

The symptoms when the disease is established may be divided up, for convenience, into those characterizing the acute attacks which so commonly occur and those persisting during the quiescent interval.

*Acute attacks* are sometimes attributable to a definite pneumonia; sometimes they represent only an acute bronchitis of the dilated tubes.

In the first case they are ushered in with fever and often with chilliness. The cough becomes hard and dry, the breathing rapid and distressed, and there is generally pain in the affected side, which is worse on coughing. Headache is complained of if the child is old enough to indicate it, and the bowels are generally costive. Vomiting may occur at the onset, or later is associated with the expectoration of phlegm. Streaks of blood may appear, or even a definite hemoptysis occur. The sputum, which has perhaps never quite ceased in the interval, now becomes excessive in quantity and may be of stale odor, or sometimes offensive. Its expectoration is preceded by attacks of violent, often spasmodic, cough at the end of which the sputum is thrown up, sometimes times with vomiting.

The child presents to observation the appearance of a case of pneumonia. The face is flushed, the breathing rapid and distressful, and he sits up in bed with an anxious expression, an expiratory grunt, and working the nose. The lips are generally somewhat cyanosed, and the whole face may present a dusky hue. The temperature is raised, perhaps

to 101° F. or more, the skin is moist or even profusely sweating, and the pulse rapid. This condition represents, indeed, a pneumonia in the already altered lung tissue and possibly in the parts around.

If the child comes under observation for the first time there may be little to suggest any other condition than a simple pneumonia, and the permanent lung disease, upon which the acute attack is grafted, may remain unsuspected until the latter has expended itself and only the signs of the former condition are left; when, however, the disease is advanced certain permanent records are at once observable, notably the chest deformity, the displacement of organs, and, perhaps, the clubbed finger-tips, and these indicate the true condition. Finger-clubbing, to be sure, does not always occur even with marked fibrosis; it was present in 16 among 38 of my own cases.

In other instances, especially where the onset has been insidious from the beginning, the acute attack may be only a slight exacerbation of the existing condition, due to acute bronchitis with congestion of the surrounding lung tissue. The symptoms are less urgent; there may be little dyspnea, no real distress or cyanosis, the alae nasi remain inactive, and the temperature is but little raised. The cough is the most troublesome symptom, and the expectoration, formerly small in quantity, is now brought up in abundance.

In advanced cases of fibrosis, especially in older children, a quite different train of symptoms may bring the child under medical care, namely, those of cardiac incompetence. The child may then show marked cyanosis, with icteric conjunctiva, and dyspnea, perhaps amounting to orthopnea. Nausea and, perhaps, vomiting are present, and there may be some edema of the extremities; in short, the case presents all the symptoms of progressive failure of the right heart. On examining the chest the cardiac dulness is found to be greatly increased, especially to the right of the sternum; at the apex the first sound is toneless and accompanied by a systolic murmur, and the heart's action is rapid and irregular. In such a case seen for the first time the lung condition may be overlooked, and mitral regurgitation due to organic lesion will probably be suspected.

In the acute pneumonic attacks first mentioned the following signs are discoverable in an advanced case.

**Physical Signs.** *Inspection.*—On examining the chest, movement is found to be imperfect on one side, generally at the base. If the condition is widespread, the whole side may appear shrunken, the shoulder and nipple lowered, the ribs closer together than usual, and the spine curved, with the concavity toward the affected side; such marked deformity is, however, uncommon. When the left side is affected visible cardiac pulsation may be observed in several spaces, owing to uncovering of the heart's surface, and this may extend out into the axilla from displacement of the heart.

*Palpation.*—On palpation the limitation of movement is more accurately estimated. The heart's apex beat is found displaced toward the shrunken lung, and the whole mediastinum drawn over. If the upper



part of the lung only is diseased, the heart's apex is tilted upward; otherwise this organ moves over as a whole with the mediastinum, its axis remaining unchanged. There is, however, in marked cases, generally some dilatation of the right auricle, and from this cause the apex tends to tilt somewhat upward to the left. The vocal vibration, where this is obtainable, corresponds roughly with the vocal resonance to be mentioned presently, but is more often diminished or absent.

*Percussion.*—Following the mediastinum the opposite lung passes over to the affected side, and its resonance may be found by percussion one-half or even one inch beyond the sternal margin. The fibroid lung gives a flat, wooden, high note to percussion with a noticeable increase of resistance, which often simulates that of fluid. The amount of dullness depends, of course, on the amount of fibrosis, but it is usually increased during the acute attacks. The percussion note in other parts of the same lung, or on the opposite side, may be somewhat hyperresonant.

*Auscultation.*—The character of the breath sounds over the affected area depends on the amount of movement in the lung, the size and nearness of the bronchiectatic cavities, and also whether these are empty or filled with secretion. The air entry may be feeble or good, but during the acute attack the quality of the breath sounds is always bronchial and sometimes amphoric. These signs may be suppressed temporarily through blocking of the tubes with secretion, but a good cough will re-establish them. The added sounds are generally bubbling, metallic, resonant râles, but sometimes the râles have a dry, rustling quality, not unlike fine friction.

The vocal resonance is increased, normal, or diminished, according to the condition present. When the lower lobes are affected it is often diminished at the base, and may be nasal or even approaching egophonic in quality, thus simulating the condition in pleural effusion; it may be absent. Higher up in the lung it is often increased and, if the cavities are large and near the surface, giving tubular or amphoric breath sounds, bronchophony or even pectoriloquy will be heard. Roughly, it may be said that where the breath sounds are merely bronchial, and especially where distant and bronchial, the vocal resonance is diminished; where the breath sounds are tubular, or amphoric, the vocal resonance is increased or bronchophonic.

*The Quiescent Period.*—When the acute attack is past the lung is left permanently damaged, but in a state of quiescence, and the symptoms depend on the stage which the process has reached.

If the condition is advanced, the signs are but little different from those present during the acute attack. The dullness may be less absolute, the tubular or bronchial breathing more distant and less marked, the vocal resonance may be diminished still more, and the number of moist sounds lessened. In less advanced cases it is sometimes astonishing how considerably the signs will diminish in the quiescent interval. This is especially the case where the bronchiectasis is the more marked feature. The signs of dilated tubes, which were present in marked degree during the attack of acute consolidation, may entirely disappear, leaving



only weak breathing with, perhaps, a little impairment to percussion.

The expectoration, also, even if abundant, may quite disappear in the intervals, and in cases where the signs of fibroid lung are marked, there may be no moist sounds audible in the quiescent stage. In other cases, on the other hand, the expectoration continues and may be brought up in large quantities at long or short intervals. The children do not waste as in phthisis, but, as a rule, are fairly well nourished, and show a healthy appetite. This is especially so in apical cases, where a suspicion of phthisis is likely to be entertained; such children are often fat and rosy.

*Position of Lesion.*—The following table shows the position of the lesion among my 38 tabulated cases:

Right lung.		Left lung.	
Apex	2	Apex	1
Base	4	Base	6
Whole	5	Whole	16
	11		22
Both lungs		5 cases.	

**Complications.**—When the lower parts of a lung are riddled with large cavities, surrounded by tough, functionless lung tissue adherent to the chest wall, it is obvious that drainage by expectoration or occasional vomiting is bound to be very imperfect. The retained secretion tends to become foul from the growth of numberless saprophytic organisms, and contains besides, in many cases, more dangerous pathogenic germs. As a result of the development of the former a septic absorption is constantly taking place and greatly undermines the health of the patient, and, as a result of the latter, the patient lives in constant danger of infection. *Empyema* not uncommonly occurs, and when recognized may be successfully treated; *bronchopneumonia* is still more common, and death not infrequently occurs in the attack. In the cases of advanced fibrosis it attacks the unaffected lung tissues and sometimes passes on to multiple *abscess* formation throughout the lung, and occasionally *gangrene*. *Cerebral abscess* is an occasional but well-recognized complication of fibrosis with bronchiectasis.

*Emphysema* is found in some cases of fibrosis, not as a compensatory process merely, but in the form of a generalized emphysema leading to the common deformity of the chest. *Cardiac disability* may be again referred to as a complication; it has already been described, under the heading of Symptoms, as a condition which occasionally brings the patient under observation. *General tuberculosis* is a fatal concomitant in some cases, but bears no true relationship to the lung fibrosis, being but an accidental complication. Lastly, *amyloid disease* of the viscera arises in some cases, but appears to be less frequent than one would be disposed to expect.

**Diagnosis.**—The diagnosis generally has to be made from one of two conditions—when at the apex, from phthisis; at the base, from pleurisy.

The diagnosis from *phthisis* may be difficult. Phthisis, it must be

remembered, is rare in children under six years of age; in them tuberculosis of the lungs takes a different form, but above the age of six years cases similar to phthisis of the adult are occasionally met with. The course of the disease, as described by the friends, often affords a valuable clue: in phthisis it is generally short, but of gradually increasing severity; in simple fibrosis it often extends over many years, the illness dating from a definite attack of pneumonia or from the bronchitis of the infectious fevers.

The signs in the lungs generally give a clue. In fibrosis the sequence of lobes so often followed in phthisis is not observed—namely, the apex of the upper lobe, the apex of the lower lobe, followed by the apex of the upper lobe on the opposite side. Moreover, in chronic phthisis of children considerable cavitation generally occurs and contrasts strongly with the more moderate dilatations occurring in apical fibrosis. The sputum must be examined for tubercle bacilli. These can be demonstrated, if care is taken, so that a negative result is of no value. The general appearance of the child is often a help to diagnosis; the subject of fibrosis generally presents a thick-featured, somewhat bloated facies, and is often well nourished, a contrast to the wasting and anemia of tuberculosis.

Basal fibrosis is easily mistaken for *pleural effusion*. Especially is it liable to be taken for empyema discharging itself through the lung, since the large quantities of green pus expectorated may closely simulate that of a purulent effusion. The signs may closely resemble those of fluid, resistant dulness, feeble breath sounds, whose bronchial quality is no bar, and, perhaps, a diminished or absent vocal resonance. There may even be something approaching to egophony. Added to these are finger-clubbing and, perhaps, the history of an antecedent pneumonia. Points may generally be found, however, to turn the balance. Thus, the position of the neighboring organs—the heart is displaced by fluid, drawn over by fibrosed lung, though even here a fallacy arises, since a chronic pleurisy may cause contraction of the chest wall and after a time draw the heart over. Signs of a cavity are a valuable distinction, as they are not found over a pleural effusion; even large bubbling râles are unlikely to occur with pleurisy. It must not be forgotten that empyema may occur as a complication of pulmonary fibrosis.

The recognition of the underlying fibrosis of the lung during an acute pneumonic attack has been referred to under the heading of Symptoms.

**Prognosis.**—This depends on the position and extent of the lesion, and the age and station in life of the patient. All the cases with grave symptoms have a basal lesion, whether the rest of the lung is affected or not; apical cases generally do well. Basal cases suffer, owing to stagnation of the secretion in the bronchial cavities; here it forms a septic focus from which may arise pneumonia, often terminating in abscess or gangrene, and pus infections, especially empyema and cerebral abscess. Foul sputum is of bad omen, as it points to retention of the secretion in the lung cavities, and the septic absorption leads to marked deterioration of health. It is often the beginning of the end.—

The more advanced the fibrosis the more marked, as a rule, the bronchiectasis, the more reduced the available lung tissue, and the greater the strain on the pulmonary circulation.

Since the lesion is irremediable, it follows that if it starts in infancy the outlook is worse than if it develops in the later years of childhood. In the poorer classes, the exigencies of life greatly increase the risks of those acute attacks which constitute its chief danger; among the well-to-do, change to a warm climate during the winter months does much to remove the risk of acute catarrhs, and the condition is consequently more likely to remain in abeyance.

**Treatment.**—The treatment resolves itself into that of the acute attacks and that of the quiescent intervals. When the acute attacks are due to a definite pneumonia, the treatment to be found under that heading will be required; when due to a bronchitis or peribronchitis, treatment will be carried out on the lines laid down for that disease. In addition, the heart condition must be carefully watched, since the fibrosis causes a constant impediment to the work of the right heart, and some incompetence is much more likely to ensue than during a simple pneumonia or bronchitis.

When the acute attack is entirely cardiac in origin, as it occasionally is, the treatment will be chiefly that employed in cases of mitral disease with loss of compensation.

In the quiescent intervals, or in the less serious exacerbations of slight and apical cases, attention must be directed above all to improving the general condition of the child. Such children can seldom with impunity stand the winter of a harsh climate, and, where circumstances permit, it is well to move them for the colder months of the year to some spot where abundant sun and still, dry air are obtainable. Under such conditions they can live much in the open, and thereby they avoid to a large extent those catarrhs which are both dangerous in themselves and also tend to further the progress of the disease. Cod-liver oil, with iron or malt, is often useful, especially if creosote be added. Guaiacol may be used instead, and a preparation I have found of the utmost value is thiocol, a creosote derivative which is alike tasteless, freely soluble, and readily borne by the weakest digestion. It may be given in doses beginning at 0.2 to 0.3 gm. (3 to 5 gr.) for children a few years old, and may be largely increased, though the small doses are often quite efficient in improving nutrition and the general well-being. It is best prescribed with syrup of orange, or syrup of iron phosphate, with or without dilution with water, but these may be omitted if they upset digestion.

When the secretion is abundant and difficult to bring up, the effect of posture may be taken advantage of in clearing the tubes; in addition, an occasional emetic may be given and a course of stimulating expectorants employed. These methods have been already described under the heading of Bronchiectasis, as has also the treatment of fetid expectoration by the inhalation of volatile antiseptics, or by creosote vapor after the method described as introduced by Dr. Arnold Chaplin.



External drainage is less applicable to cases of pulmonary fibrosis than to simple bronchiectasis, owing to inability of the surrounding parts to fall in and close the discharging cavity.

### FOREIGN BODIES IN THE AIR TUBES.

The entrance of a foreign body into the air tubes is an accident of not very rare occurrence in young children. The objects inspired have been very various, and include such examples as a glass bead, a pill, a bean or seed, the peg of a top, a fruit-stone or grain of corn, and a bone from soup. These were the foreign bodies found in a series of cases. In addition, a caseous lymph node may ulcerate into a bronchus and cause blocking of its lumen.

**Symptomatology.**—A common history is that the child is playing with his toys, or otherwise amusing himself, when he is suddenly seized with a violent fit of coughing and choking, during which he turns purple in the face and gasps for breath. The attack lasts a variable period, sometimes as much as fifteen minutes, and then passes off, and the child may be quite comfortable for a time, but generally a second similar attack occurs after an interval. These attacks of coughing may be repeated indefinitely, the child being comfortable in the interval, and he is sometimes thought to be suffering with whooping-cough, which the paroxysms may closely simulate even to the accompanying "whoop." In cases where the body becomes immediately impacted, no recurrence of the initial attack may occur.

Occasionally the foreign substance is expelled during an attack; more often it remains. The position of the impaction in the tubes and the results of its presence there depend upon its size, shape, and consistency.

If large, it may block the larynx and lead to immediate death, occasionally occurs from impaction of a lump of meat. If small, it may become lodged in the ventricles of the larynx, leading to symptoms resembling laryngitis stridulosa, but more commonly it enters a bronchus generally the right, and either remains loose, when it is coughed up against the vocal cords and causes attacks of spasm, or becomes impacted in the tube. In the first case it may be heard to move up and down during coughing, and, if the larynx is palpated, the vibration of its impact on the vocal cords may be distinctly appreciated. When it is impacted in the bronchus, the symptoms will depend on its shape. If it is spherical, like a bead or bean, it may completely block the tube. In this case the air entry and respiratory movement will cease over the affected part, which may be the whole lung or only one lobe, the blocked area will collapse, and the heart and mediastinum move over toward it. If the body is irregular in shape, and does not obstruct the passage of air, no pulmonary collapse results, but ulceration is set up by its pressure, followed by an acute bronchitis of the tubes below.

In either case the outlook is now very serious; a septic bronchopneumonia, or abscess or gangrene of the lung may be set up, especially



where the foreign body is of a nature to undergo decomposition, but in more favorable cases bronchiectasis of the tubes below the obstruction takes place, the lung becomes fibrosed, and the abundant secretion retained in the dilated tubes generally becomes fetid.

In some cases the presence of a foreign body is quite unsuspected, the child being brought some years after the accident on account of the expectoration of foul pus. On examination a unilateral fibrosed lung with bronchiectasis is found, and by questioning the parents a history of whooping-cough at the onset may be elicited. This seems to agree well with the etiology of a simple fibrosis of the lung, and it may well be overlooked that the so-called "whooping-cough" represented in reality the spasmodic attacks set up by the foreign body before it became impacted. In some such cases an empyema appears outside the fibroid lung, and instances are recorded where a superficial abscess in connection with the foreign body has formed over the chest wall.

The *physical signs* are not peculiar to the presence of a foreign body, but vary according to the condition it sets up. When a main bronchus or large division is completely blocked, at first the lung is resonant, but the respiratory movement is lost, and the breath sounds absent. In some cases the body may become loosened by cough and the normal signs reappear for a moment to disappear again presently. This is pathognomonic of the presence of a foreign body in the bronchus. After a time the imprisoned air in the lung is absorbed, it collapses, the percussion note becomes impaired or dull over it, and the heart and mediastinum move over toward the affected side. Where the bronchus is not completely blocked, the air entry may be poor over the lobe or lobes connected with it, and there are found the signs of bronchitis localized to the affected parts, these last appearing very rapidly after the onset.

When bronchopneumonia or pulmonary abscess supervenes the signs belonging to those diseases will be present. If bronchiectasis is set up, the symptoms of this disease generally appear in a few weeks' time, though the sputum may not become foul for a year or two after the onset in cases where the foreign body is smooth and clean; where it is of an irritating nature, or capable of decomposition, the sputa become rapidly fetid. In these cases the physical signs are those of unilateral bronchiectasis and fibrosis, and the reader is referred back to the description of these diseases.

**Diagnosis.**—This depends upon the sudden onset of symptoms of choking in a healthy child, followed by the signs and symptoms described above, and in some cases the nature of the article is known or suspected. When the body is loose in the tubes its movements may be heard or palpated. Where the symptoms are those of laryngitis the history of onset will generally determine the diagnosis. In cases where a so-called "fit" has occurred during the progress of a meal, and the patient is found to be half asphyxiated or unconscious, the larynx should be at once explored with the finger on the suspicion of food impaction. Where

a unilateral bronchiectasis and fibrosis are found, especially if the expectoration is fetid, the symptoms of its onset should be carefully investigated, and the nature of any initial, so-called "whooping-cough" ascertained. Examination by x-rays will in some instances lead to a correct diagnosis.

**Prognosis.**—The prognosis is bad if the foreign body remains in the tubes, though there is a chance that it may become loosened and be removed by cough. If this does not happen a fatal issue must be expected, though an interval of many years may pass when the foreign body is some clean and smooth article such as a glass bead or the peg of a top. In cases where sharp and angular bodies are impacted, especially when liable to decomposition, as was the soup-bone cited above, the outlook is very unfavorable. Septic trouble is liable to intervene and lead to death. When the foreign body is removed the prognosis depends on the amount of permanent damage left behind, but even considerable bronchiectasis is compatible with good general health.

**Treatment.**—At the onset the patient should be inverted and shaken. This is not often successful, as the object can seldom pass the glottis, and urgent laryngeal spasm may be set up. When the presence of a foreign body is decided upon, immediate steps for its removal must be taken. Tracheotomy should be performed and the child again inverted and shaken; this is generally successful; if it is not the wound must be kept open, the edges being retracted with the aid of an elastic band round the back of the neck. By this means the body will be coughed out through the opening if it becomes loosened, though sometimes it passes through the glottis and is swallowed. If it remains, after an interval an attempt may be made to grasp it with a fine forceps if its presence is undoubted. It will usually be found at the bifurcation of the trachea, or in one or other bronchus, generally the right on account of its larger size, and in the inclination of the dividing spur to the left side.

In cases where the patient is first seen after bronchiectasis has been set up the necessity for operative measures must be discussed, since the condition is certainly fatal if the foreign body remain. Having localized the position of a large dilated tube, a piece of rib is resected at a chosen spot over it, and the lung stitched to the wound unless adhesions between the pleural surfaces already exist. After a few days the bronchiectatic cavity is opened with a Paquelin cautery at dull-red heat, and an attempt made to find the foreign body. This is, of course, impacted above the cavity, and if it is not found a large drainage tube must be left in, since it is sometimes expelled through the wound subsequently during coughing. When the operation is unsuccessful, it is recommended to perform tracheotomy and explore the bronchi, or to open the pleural cavity elsewhere and examine the surface of the lung with the finger. When the foreign body is found and removed the general health may be largely or entirely regained, the expectoration becomes less in quantity and loses its fetor, and the case becomes one of ordinary basal bronchiectasis and fibrosis, whose treatment has been already considered.

## SECTION VIII.

### DISEASES OF THE HEART AND BLOODVESSELS.

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#### CHAPTER XXVIII.

##### METHOD OF EXAMINATION—CONGENITAL HEART DISEASE— RHEUMATIC HEART DISEASE.

##### THE CLINICAL EXAMINATION OF CHILDREN WITH HEART DISEASE.

THE examination of a child must be methodical. Only by this means can rapidity and accuracy of diagnosis be eventually achieved. This accuracy is the more needed because heart disease in the child is much more common than the general public believe, and the symptoms are not always appreciated by the medical man in attendance.

The aspect of the face will naturally first attract attention. Is it flushed with a purple tinge as in mitral stenosis? is it pale as in aortic regurgitation? is it distressed as in pericarditis, or blue as it is in congenital disease?

The physician will soon see if the breath is short, and should not mistake the way in which such children spare their words for taciturnity. He will look at the hands, note the color of the nails and the shape of the tips of the fingers, and then examine the pulse.

**The Pulse.**—This should not be described in a loose way as soft or hard, but under these headings:

1. Rate. 2. Regularity in force and frequency. 3. Character of the wave: (a) Well or ill sustained. (b) Size, large or small. (c) Compressibility. (d) Felt or not, between the beats. 4. The condition of the arterial wall. 5. Any special peculiarities.

The *chest* is now examined. First the heart, by inspection, palpation, percussion, and auscultation, and, if necessary, by radiography. After the heart the lungs will be investigated, and the vessels in the neck can be observed.

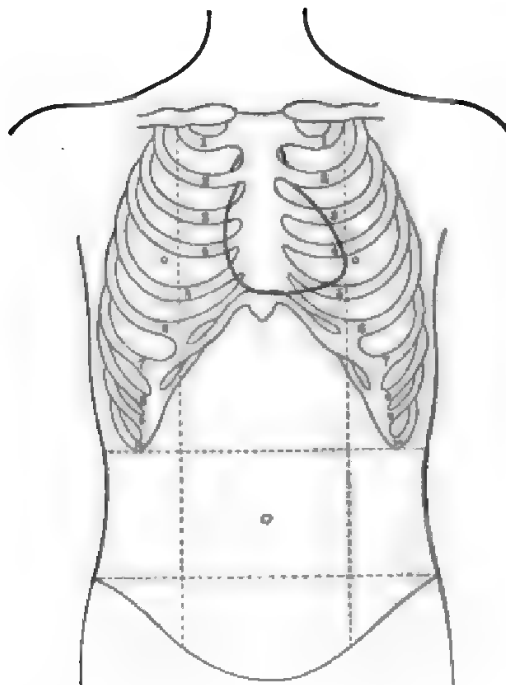
The *abdominal organs* are next investigated and three special points are noted: 1. The condition of the liver. 2. The condition of the spleen. 3. The presence or not of ascites.

The lower extremities are examined to settle the question of edema, and then other points which have bearing upon the case are attended to, as, for example, the presence of nodules or arthritis, or the slight incoordination of an early chorea.

The urine is always to be tested.

Lastly, it is an excellent plan to have a stamp outline of the chest and abdomen on which the chief results of this examination can be recorded (Fig. 143).

FIG. 143



Stamp outline used in making notes in heart disease.

**The Position of the Heart in Childhood.**—The heart of the child lies higher in the chest than that of the adult, and the precordial area is more variable.

The apex impulse in the infant is often indeterminable. Up to about six years it is situated in the fourth intercostal space; after seven, in the fifth. The area of relative dulness always reaches, as in the adult, to the right of the sternal margin, but it extends farther to the left, reaching the vertical nipple line, and even passing beyond it in the first six years of life. Under six years of age the upper limit is usually at the second interspace to the left of the sternum, and after six years the third rib.

The relative dulness is the important area of dulness and needs careful light percussion for its determination.

I believe in the finger as a pleximeter, and though I am convinced



the very great importance of careful percussion, it is, in my experience, but a partial assistance. To draw important conclusions from minute differences in the percussion outline of the heart is dangerous. In no disease more than in those of the heart in childhood is it more necessary to take a broad survey of the case, and to steadfastly refuse to be led to forming one's judgment from one physical sign alone, be it an area of cardiac dulness or a bruit. Symptoms in heart disease are ten times more important than physical signs, and this truth, impressed upon me by my honored teacher, Sir William Broadbent, is a very great one.

#### HEART DISEASE IN CHILDHOOD.

**Some Peculiarities in the Heart Disease of Childhood.**—When, in general terms, a comparison is drawn between affections of the heart in the child and in the adult, certain differences are apparent. In the child the symptoms are often remarkably latent, while breathlessness in a child means more than the same in an adult. Wasting is a more prominent symptom in the child. Children have less pain and anxiety, except in diphtheria, sudden death is extremely rare. Great edema is exceptional, but slight edema of the face is more common than in the adult. The pulse is a more uncertain guide in the child, for it is more easily influenced by fright and nervousness. Pericarditis is more common, and the lesions of endocarditis are often multiple. The heart, as a whole, is more often damaged, and carditis or a relapsing carditis is much more frequent.

Hypertrophy is rapidly and effectually accomplished, but the greater liability to repeated attacks of rheumatism leads to frequent breakdowns in compensation, which often stultify the value of the hypertrophy. A child with advanced valvular disease is much less of an invalid than an adult. The frequency, however, of pericarditis and the occurrence of pericardial adhesion in early life add to the difficulties with which the physician has to contend. Degenerative affections of the heart and blood-vessels are practically non-existent; nor does overstrain play the part of causation that it does in adult life. On the other hand, infections are much more liable to cause heart disease in the young.

**Etiology. GENERAL CONSIDERATIONS.**—In considering the etiology of heart disease in childhood it is usual to divide the subject into two great groups: 1. Congenital heart disease. 2. Acquired heart disease. This is a clinical division of practical value, though it is apparent on closer inquiry that the distinction is not always a scientific one.

1. *Congenital Heart Affections.*—These are in part the result of arrest of development of an organ, which is gradually evolved from a simple tube into a complicated four-chambered pump, supplying an intricate system of vessels, and partly the result of disease attacking this organ through the medium of the placental circulation of the mother.

2. *Acquired Forms of Heart Disease.*—It is an encouraging fact that the causes of acquired heart disease are generally definite ones. The greatest proportion of them result from infective processes. This fact must

make a writer upon this subject long for an inspired pen to arouse in his readers an enthusiastic desire to study their prophylaxis. With the demonstration of the infective nature of rheumatic fever much of the obscurity that hitherto existed has faded away, and the task of giving a clear description of the subject is greatly lightened.

*Rheumatism* is the chief cause of acquired heart disease, the channel of infection in many cases being through the inflamed tonsils. A history of rheumatism following a sore throat is very common, a fact that for over a century has been recognized by clinicians. In 1900 Dr. Paine and I produced the lesions of rheumatic fever in rabbits by subcutaneous inoculations with a diplococcus isolated from a case of rheumatic angina. This micro-organism we had previously isolated and demonstrated in the chief lesions of rheumatic fever.

Meyer independently arrived at the same conclusion by a study of rheumatic angina, and it seems certain that the tonsil is a channel of infection. It is disputed whether one or various infections may cause rheumatic fever, but at the present time there is, in my opinion, no positive proof of the existence of more than one microbic agent—a diplococcus of the streptococcal group—a fact previously suspected by many observers. Rheumatic fever is, no doubt, more common and more virulent in England than in America, and for this reason is a more important factor in the causation of heart disease in the former country. In childhood it is peculiarly liable to attack the heart, and the physician should not overlook the danger from mild attacks of rheumatism.

*Scarlet fever* is another important cause, but the nature of the infection of scarlet fever is much disputed; and though some hold it to be the result of a streptococcal invasion, others look upon the damage to the heart either as truly rheumatic or in other cases as being due to a streptococcal infection which is a complication and not the true cause of the scarlet fever.

*Diphtheria* damages the heart in a considerable proportion of cases by the action of its poisons upon the myocardium, rather than from the deposition of the bacilli in the valves and pericardium.

*Tuberculous infection* is another factor, but not a very common one, and perhaps more frequently met with in America than in England.

*Influenzal infection* attacks the heart, and, although it is the elderly who are the chief sufferers, still there is clear evidence that children may also, to a lesser degree, be damaged by this effect of the disease.

The *pneumococcus*, *streptococcus pyogenes*, *gonococcus*, *meningococcus*, and *staphylococcus aureus*, singly or in mixed infection, occasionally attack the heart, but the frequency of the occurrence will not compare with that of the rheumatic infection.

There are examples of heart disease after *measles*, *chickenpox*, *typhoid fever*, and *pertussis*, and, in some cases, *congenital syphilis* is claimed as a cause.

PREDISPOSING CAUSES.—Enough has been written to show what a prominent part infections play in the causation of heart disease, but the predisposing causes are also of great importance.

Among them *heredity* stands first, for it is a powerful factor in rheumatism. The *seasons of the year* in which heart disease is most likely to commence are the autumn and the spring.

Damp, and especially cold damp, overcrowding, and malsanitation all lower vitality and predispose to tonsillar inflammations. Carious teeth, with alveolar inflammation, chronic otitis media, and other chronic discharges are all sources of danger which may permit infection by bacteria that have the power to produce heart disease, but, as the late Dr. Packard pointed out, it would be a mistake to accept every infection of the heart entering from the mouth or upper air passages as necessarily rheumatic in nature.

When *renal disease* is considered, the questions involved are more complex. The heart is often damaged, and the damage may occur in two different ways. A child may fall ill with nephritis, peritonitis, and pericarditis, a result, let us suppose, of a pneumococcal infection; in this case the nephritis and pericarditis are results of this infection, and the pericarditis is not dependent upon the nephritis. Again, in rheumatism the diplococcus can be found in the kidney and may cause nephritis as well as carditis. But there is also in renal disease, especially of the chronic type, a retention of poisons which produce important effects upon the heart and bloodvessels, and it is this group of cases which is especially associated with renal disease. Nevertheless, the subject is obscured by the occasional supervention of an acute, infective pericarditis, even in these chronic cases.

*Overstrain* will damage the hearts of young, anemic, rapidly growing children. When a child is healthy it takes a great deal to damage this organ, and it is very necessary not to get an exaggerated idea of the influence of overstrain. It will be an evil day when the heart specialist, who has never perhaps been young himself, undertakes to arrange the exercise of a healthy boy. But where there is imperfect convalescence from some acute infection, such as influenza or diphtheria, then there is danger. Underfed and growing lads, who bicycle about as messengers and errand boys, or who train for races after a hard day's work, are liable to strain the heart; as also will delicate, high-spirited boys who are sent for long runs or made to exercise beyond their strength.

Where there is already organic heart disease, the influence of overstrain is a much more dangerous one and may bring about a very serious breakdown. Thus, at a time when bicycling was the madness of the hour in this country, a boy with advanced mitral stenosis went for a long ride upon a hot day; the result was that he collapsed with extreme tachycardia from which he took months to recover, and came very near, indeed, to losing his life.

*Anemia* in childhood intensifies the effect of any cause which produces organic heart disease, and when it is very profound enfeebles the cardiac muscle to such a degree that it becomes a danger in itself. More often, perhaps, by the production of loud functional murmurs, it causes difficulty by raising the question whether these murmurs are not in reality organic and dependent either upon congenital or acquired heart disease.



*Nervous influences* are important in the clinical study of heart disease in childhood. *Chorea*, I look upon as, in most instances, if not in all, rheumatic in origin, and in this article the affections of the heart which occur with chorea are considered under the heading of Rheumatism. But it would be an error to lose sight of the detrimental effects of shock, fright, and evil habits upon the heart. Irregularity of action, palpitation and rapidity, are common results of these influences, and a sudden fright may cause even fatal syncope.

*Pulmonary affections*, as, for example, asthma or repeated bronchitis with emphysema, may, even in childhood, so greatly tax the right side of the heart as to cause tricuspid incompetence.

*Digestive disturbances* also cause functional cardiac disturbances, and in small and weakly infants gastric distention may so embarrass the heart as to cause death.

It is difficult to write in any precise terms concerning the influences on the heart of rapid growth and development about the time of puberty, but these tax a damaged heart and predispose to a breakdown unless particular care is bestowed upon children at that age.

Lastly, there are mysterious causes of heart disease which are met with from time to time. The best example is a curious group associated with an enlarged *thymus*. These cases, fortunately very rare, are highly dangerous, and fatal syncope may occur. As to whether the condition of the thymus has any causal relation to the cardiac failure is still disputed, but the association of the two rests on sound clinical observation.

Even more rare are cases of suprarenal hemorrhage in infancy, which may lead to rapid cardiac failure and death. This, however, is mentioned in the article on Diseases of the Suprarenal, *q. v.*

#### CONGENITAL HEART DISEASE.

There are two main groups of congenital heart disease: (1) one in which malformation occurs; (2) the other in which there is intra-uterine inflammation of the valves, with secondary defects resulting therefrom (Fig. 144.)

1. In the first group the arrest of development may take place in early fetal life, and the heart only consist of two cavities, a ventricle and an auricle, with a single vessel for the pulmonary and systemic circulation. In other cases there are two auricles and one ventricle.

If the arrest is at a later period, then the septa between the auricles and ventricles are imperfect and the aorta and pulmonary artery only partially developed. Or, again, the large vessels may be displaced.

In the later period of fetal life it is sometimes difficult to decide whether the imperfections in structure are due to disease or malformation, and thus these two groups overlap one another, but it is in the later days that premature closure of the foramen ovale, or premature obliteration of the ductus arteriosus occurs.

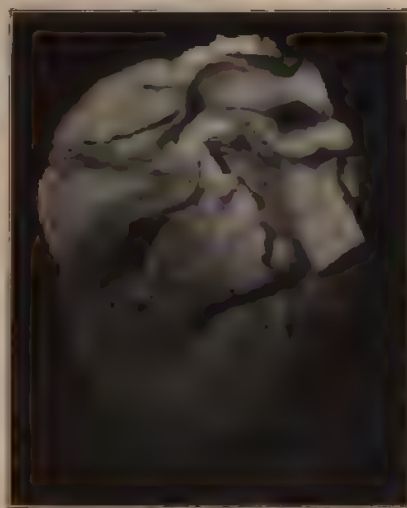


2. The second group is of more interest; here are found those very remarkable cases of rheumatic fetal endocarditis of which the following, from the museum catalogue of University College Hospital, is a good instance:

A child, who lived for thirty days, was discovered the day after birth to have a loud systolic murmur, heard all over the precordium and at the back. It was a small, quiet, and pale infant, but except for occasional blueness of the lower eyelids showed no cyanosis. The mother had suffered twice from rheumatic fever, and was attacked a third time during her pregnancy. Her three other children were healthy.

The necropsy showed that the interventricular septum was deficient at the upper and back part, and to the margins of the aperture the cusps

FIG 144



Congenital endocarditis of pulmonary valve. Congenital heart disease. The pulmonary valve is exposed and shows endocarditis.

of the mitral and one cusp of the tricuspid valve were adherent. Both valves were beaded by vegetations. The foramen ovale was patent, as also the ductus arteriosus. The other valves were natural.

Another striking example, which came under my own observation, was that of a child born cyanosed and in an almost asphyxiated condition. Death followed on the third day. During pregnancy the mother had suffered from a severe attack of rheumatic fever. The necropsy showed extensive and recent mitral endocarditis.

The diagnosis of fetal endocarditis has been made even in uterine life, as in cases reported by Peters and others.

In the second group, then, the important lesions are connected with fetal endocarditis and anomalies of the valves and cardiac septa.

The most important *clinical* cases in this group are those in which

there is narrowing of the pulmonary artery. This is the form of congenital heart disease in which life is far more likely to be prolonged to adult age than in any other.

There is frequently associated with this stenosis a patent inter-ventricular septum: the opening is, as a rule, a small one at the upper part of the ventricular septum, in the membranous, undefended space. In addition, the foramen ovale may be patent and the ductus arteriosus incompletely closed.

Stenosis of the aortic or mitral valves is also met with occasionally, but the reader must be referred to standard works upon the subject for further details.

**Symptomatology.**—When well marked, the symptoms of congenital heart disease are very striking ones, and when, in spite of them, a child survives infancy, his appearance is often so characteristic as to leave an indelible impression on the memory. It is simple and useful to consider the symptoms first as they occur in infancy, and afterward as they occur in older children. This, too, is justified by the fact that the majority of cases die when under two years of age.

These infants are, as a rule, quiet and listless; often small and puny. The nature of the complaint may be detected at once, or it may not show itself or be overlooked for some months. *Cyanosis* is the symptom which most attracts attention, and this is intensified whenever the infant cries, and is generally detected by the mother or nurse. It should be borne in mind that cyanosis is not always present, and in other cases is so slight as to escape notice; thus it is not unusual for the medical attendant to discover the condition of the heart when going through a routine examination of the child, which has been called for by its failure to make progress and by its general feebleness. This cyanosis implicates both the skin and mucous membranes, and when it is extreme reaches a mulberry hue. The explanation of its occurrence has not been agreed upon. Some have attached great importance to venous congestion; others more importance to deficient aeration of the blood. Of late a good deal of attention has been directed to the increase in the number of red blood corpuscles which have been found in these conditions. This is not peculiar to congenital heart disease, but it is sometimes very well marked in such cases, and was well described by Toennissen. A case examined by Baumboltzer gave the following result, viz. red blood corpuscles, 9,447,000; specific gravity, 1071; hemoglobin 160 per cent. Thus, there is not only increase in the number of red blood corpuscles, but a concentration of the blood itself, which tends to make its passage in the vessels more difficult.

Cyanosis, although the most important, because the most frequent symptom—hence the name *morbis ceruleus*—is by no means the only index of congenital heart disease. In addition such children have cold extremities, and in some cases labored respiration, or paroxysms of disordered breathing, with unconsciousness and epileptiform attacks. These cerebral attacks are sometimes prolonged and most dangerous. In other cases the rapid action of the heart may attract the mother's

attention, and for this symptom alone a child may be brought to the doctor and a congenital malformation discovered.

Clubbing of the fingers, toes, and nose is not so frequent as cyanosis, but whenever there is any suspicion of congenital heart disease it should be looked for. It may occur with or without cyanosis, but, as a rule, is rather later in its appearance.

On *physical examination* the question of diagnosis is usually settled. The heart is found slightly enlarged, especially to the right of the sternum, and there is sometimes bulging of the precordial region. On auscultation a loud systolic murmur is audible over the precordium, with its point of maximum intensity over the pulmonary artery immediately to the left of the sternum. This bruit is harsh and dominates all other sounds, and is by far the most important sign of congenital heart disease. The observer may be struck by the rapidity of the action of the heart, even in an infant, and the pulse on slight exertion becomes feeble and irregular. Lastly, a fine systolic thrill can be detected by the hand placed over the upper part of the chest.

This is a description of the ordinary bruit of moderate pulmonary stenosis. The bruits, however, are not all of them loud, but may be soft and whiffing, and then should the child cry they are easily overlooked. In such cases of doubt it is very wise to insist upon more than one careful examination before pinning one's self to a definite opinion.

Cases which are still more puzzling are those in which, during the first few weeks, there is no bruit, although there are the symptoms of congenital heart disease, and yet which later develop a loud bruit, with a diminution in the urgency of the symptoms. The bruit is not always basal, or heard at its loudest at the base of the heart to the left of the sternum; sometimes there is an apical murmur, and in other cases the murmur may be diastolic. Yet, again, another bruit may cause a continuous humming sound throughout the cardiac cycle, and when this is heard at its loudest to the left of the sternum it suggests a patent ductus arteriosus. The second sound, then, is sometimes noticed to be singularly loud and clanging, and after death a dilatation of this pervious ductus arteriosus has been discovered. Under normal circumstances the ductus arteriosus is closed within the first fortnight of life.

The lives of these infants are very precarious. Sometimes they die quite suddenly, an occurrence which is so alarming and distressing that it is well for the medical attendant to bear it in mind when treating such cases. The temperature is often low, and the least exposure to cold may result in an attack of bronchitis. An attack of gastroenteritis, or of measles, or any other infective disease may prove rapidly fatal, and thus it is that either from the severity of the cardiac lesion itself or from some complication superadded to it many cases die in infancy. Yet there are a considerable number of children who survive, and among them are found the most classical examples of congenital heart disease.

Since the lesion in these cases is, as a rule, some degree of pulmonary stenosis, the physician will probably find that, in addition to cyanosis



and clubbing of the fingers, there are a cardiac area increased to the right, a systolic bruit audible in the second interspace to the left of the sternum, a faint pulmonary second sound, and a long systolic murmur, the maximum intensity of which is at the base.

The amount of hypertrophy varies considerably, if one can formulate any general rule; it is that the enlargement of the heart is often surprisingly slight. Yet there are undoubtedly cases in which the breadth of the cardiac dulness is extraordinarily increased.

A very interesting result in some cases is an arrest of development. Two cases of my own, both of them little girls, exemplified this well, one of whom resembled a doll; her features, bones, and muscles were all small and delicate. These children were intelligent, although not strong enough to undertake any sustained mental effort. There are other cases in which the intelligence is deficient, and a special allusion must be made to the occurrence of the Mongolian type of imbecility in association with congenital heart disease, as was pointed out by Garrod. This is a very serious matter, for such children are not only short-lived, but, even if they survive, are never able to earn a living. There are other cases in which the frame is not stunted by this condition; the child may be both stout and strongly built. The older children, just as the infants, feel the cold very much, and prefer to sit hugging the fire, for exertion makes them short of breath. One such patient had a great weakness for drinking hot beer, which his father, who kept a public house, prescribed for him on his own responsibility. Although the very fact that these children have survived infancy is proof that they have some vitality, they have also great dangers to contend against. The development of the body with the commencement of puberty throws a strain upon them and tuberculosis is more apt to attack them then, or later in life, than before puberty. It has been my experience to find that between the ages of two and twelve an attack of endocarditis has been the most frequent cause of death, but of the importance of tuberculosis there can be no doubt, as was clearly demonstrated by Peacock.

The development of a chronic cough and a history of wasting would put us on our guard against tuberculosis, but no hurried conclusion should be drawn. The naturally blue color of the faces of these children may lead to an exaggerated idea of the gravity of the intercurrent pulmonary disease, and the medical man be led to make somewhat hastily a most gloomy prognosis.

Other respiratory affections, notably bronchitis and pneumonia, are serious occurrences, because of the extra strain they throw upon the already impaired right ventricle.

The development of endocarditis is a very important complication and by no means easy to detect, for it will be readily understood that with a loud murmur due to the malformation already present, one of the great proofs of a recent endocarditis, the development of a new murmur is liable to be obscured. The occurrence is most serious, for the endocarditis is usually malignant in type.

In some cases it is an evidence of an attack of rheumatic fever, for



pericarditis and arthritis may occur simultaneously; in others the cause is obscure and spoken of as infective. The fever may be high and irregular, and there is increased dyspnea and precordial pain. The action of the heart is much excited, and generally it is possible to get so far in the diagnosis as to recognize that there is some acute complication causing these serious symptoms. If a bruit of recent origin, and localized to some other valve, can be detected, as, for example, an aortic diastolic murmur or a mitral systolic, the significance is very great, and this will be the most reliable direct evidence.

Pericarditis and other manifestations of rheumatic fever are also valuable aids in determining the presence of acquired disease.

It is not surprising to meet with this complication, for it is recognized that some cases of congenital heart disease are due to intrauterine rheumatic endocarditis, and the recent attack is but an exemplification of the well-known tendency of rheumatic children to be again and again attacked by rheumatism.

Another danger to life is a gradual failure of compensation, comparable to the failure which is seen so often in acquired heart disease. On the whole, this is less common than perhaps might have been expected. The right ventricle dilates and the tricuspid valve becomes incompetent, and then there follow the usual sequence of events—dropsy, ascites, congestion of the lungs, engorgement of the liver, and albuminuria.

In such cases it is sometimes difficult to determine whether all this **has** not been really the result of acquired and not congenital heart disease. When there is no very definite history to serve as a guide, **and** when for some reason or other the upper lobe of the left lung **has** retracted from the cardiac area and exposed the pulmonary artery, **it** is sometimes most difficult to decide between congenital and acquired disease, for a hemic pulmonary murmur, when the upper lobe of the left lung is thus retracted, may be so greatly intensified as to very closely resemble a congenital bruit.

**Diagnosis.**—This is not, as a rule, difficult, and when difficulties **arise** they depend either upon the absence of cyanosis or of a reliable **history**.

In the absence of cyanosis the condition may be quite overlooked, **and** without any reliable history it may be thought that the disease is **acquired**. When both congenital and acquired disease of the valves **are** present, the twofold nature of the lesions may not be recognized, **alt**hough, even if this should be the case, this may prove to be rather of **academic** than practical interest.

Great dilatation and hypertrophy of the heart suggest acquired disease, as also do apical, systolic, and diastolic murmurs. Cyanosis may result from emphysema, advanced tuberculous disease, or mediastinal growths, but, as a rule, the bruits of the congenital affection prevent any mistake. Some cretins are remarkably cyanosed; this affects the extremities, and on more than one occasion I have heard considerable doubt expressed as to whether the condition of cretinism could really explain this phenomenon. In such cases treatment by thyroid extract

has settled the question, and the cyanosis has rapidly disappeared with the improvement in the cretinous symptoms.

The details of the *differential diagnosis* of the various forms of malformation are beyond the scope of this article, and, moreover, unsatisfactory. We may go hopelessly wrong in such attempts, and find after death a condition utterly different from that which had been surmised during life. Some of the main indications are given under the symptoms of the disease.

**Prognosis.**—The general prognosis, since it includes every sort of malformation compatible with live-birth, is grave. The first principle is to reckon symptoms as more important than physical signs. A small opening in the foramen ovale or in the septum between the ventricles may give rise to no symptoms at all. On the other hand, such symptoms as paroxysms of dyspnea, or convulsions, or a persistent low temperature are very ill omened.

Another important point in the general prognosis is the social status of the patient. A child who can be given all the advantages of a warm climate, and can escape in after years the not unmixed blessing of having to earn a living; who can be well clothed, and be educated by tutors, stands a far better chance than the child who sells matches in the streets, with icy cold extremities and no proper meals.

The occurrence of an attack of acute rheumatism is an exceedingly serious matter, and liable to end in a malignant endocarditis. Repeated bronchitis, pneumonia, tuberculosis, and all acute maladies, including influenza, may entirely alter the prognosis in a case apparently favorable.

When the condition of the heart itself is taken into account, the prognosis is better in lesions of the ordinary type than when the lesion is an unusual one. It is not indeed possible, even with the ordinary systolic bruit and thrill, to be absolutely certain of the nature of the malformation, but in general it means a pulmonary stenosis, and this if moderate in degree, is compatible with a life reaching to adult years.

Laurence Humphry points out that the prognosis is better when with pulmonary stenosis there is an opening in the ventricular septum for this opening eases the pressure in the right ventricle.

A systolic murmur heard at its maximum intensity about the middle of the precordial area, and not giving rise to a thrill or to hypertrophy of the right ventricle, suggests this particular lesion of a patent septum ventriculorum.

The extent of the cardiac dulness to the right of the sternum is also some guide, for when the increase is considerable, either the lesion is considerable, or the strain on the right side is great in proportion to the extent of lesion.

The degree of cyanosis cannot be relied upon in the question of prognosis. The most cyanosed cases that I have met with have been children over eight years of age. On the other hand, it happens by no means uncommonly that infants with congenital heart disease and little or no cyanosis die quite suddenly. The only warning that may be given

in such cases is a refusal to take food and a general surface coldness. Again, it does not follow that because there is only a slight degree of cyanosis, which has only been observed when the child began to walk, there may not also be a rapid development of serious symptoms and death.

Cyanosis, in its most marked degree, is associated with such lesions as pulmonary stenosis and patent septa, and these are recognized as the less severe types of malformation.

In most cases a fairly accurate idea of the future can be obtained by keeping in mind these facts, and with caution the parents can be prepared to see the true meaning of such a serious malformation. For my own part, I am not a believer in attempts at dramatic prognosis, and feel that to assume an attitude of certainty, where there is so much uncertainty, is only to tempt fate. To say "He will die in three months," and to be correct, is, at best, to win a gloomy triumph, and if the patient lives as many years, the doctor becomes an object of ridicule. Some cases live on to thirty, forty, or even sixty years of age.

**Treatment.**—Treatment is palliative. These children must be kept warm and very carefully clothed with this in view. If possible, they should always live in a warm and equable climate. I prefer to give them an excess of fat, if they can digest it, and I also attempt to keep them fat. Whenever possible, they should be educated, but no mental strain should be permitted. In the event of their living, their employment must be light, and they should have plenty of sun and fresh air. The exercise allowed must be adapted to each particular case, and will always need caution. Cod-liver oil with iron or malt are useful prescriptions. Digitalis is not so useful as strychnine as a cardiac stimulant. When there is an attack of heart-failure with great lividity the application of leeches is indicated to relieve the veins of blood. For the fainting attacks I have often found exceedingly useful a prescription of sal volatile (carbonate of ammonium), ether and peppermint.

The general rules for the treatment of acquired heart disease are equally applicable to this affection.

#### RHEUMATIC HEART DISEASE, INCLUDING THE HEART DISEASE OF CHOREA.

Acute rheumatism is the most frequent cause of heart disease in childhood, at which time it is more liable to damage the heart than later in life. During the first three or four years of life, however, rheumatic fever and the consequent heart disease are rare. The explanation of this, so far as the poor are concerned, lies probably in the fact that the very young have not the same amount of exposure to cold and wet; nor is there the influence of school life, with its crowded rooms, foul air, and journeys to and fro, often made on a stomach not too well filled.

The incidence rises steadily from four years of age, and about ten



reaches its maximum, though for some years after it is frequent enough. As rheumatism is most rife in spring and autumn, so, too, the frequency of this form of heart disease rises at those times; there is also a greater tendency to heart affections in some years than in others.

Statistics as to the relative frequency of its occurrence in rheumatism have now, I think, served their purpose, and have shown that every case of rheumatism in childhood should be looked upon as a probable cause of heart disease.

In regard to the influence of heredity it has long been admitted that there is a family tendency to heart affections, as there is also to renal or nervous ones. So far as this form is concerned it is explained by the fact that the rheumatic predisposition is strongly hereditary. The heart disease is, in my opinion, a direct effect of the rheumatic infection and not a complication dependent upon some secondary process.

To me, then, rheumatic heart disease is a direct consequence of the access of the infective agent of rheumatism to the cardiac valves, the pericardium, and the heart wall through the channels of the coronary bloodvessels. The lesions are the results of the poisons of the bacteria, and of the vital reaction of the tissues to those poisons. These lesions, I further hold, are specific lesions, though the reader should clearly understand that there are many who would dissent from this; and although in agreement up to that point would here differ, and hold that many different infections may cause rheumatic fever, and, therefore, rheumatic heart disease.

In addition to the causes already mentioned, namely, age, heredity, and the season of the year, there can be little doubt, I think, that a cold, inclement climate, a clay soil, and damp houses are also factors, and must be taken into account. Overcrowding and malsanitation would also appear important, for rheumatic heart disease is especially common in large towns. The path of infection, so often by way of the tonsils, points also to the congregation of children in schools as a factor.

It is, I believe, an important matter to reconsider the predisposing causes of rheumatic heart disease by the light of the infective nature of rheumatism, and I trust that the medical profession will soon make some great effort in this direction on behalf of the children of the poor. Personally, I attach no importance to diet, beyond considering that a gross error, such as giving large quantities of meat to the young, is detrimental to their general health.

As rheumatic heart disease is the most frequent and most important of all heart affections, and one of the most important subjects in children's diseases, it will be made in this article the pivot upon which a description of all the other acquired forms will turn, for the same general principles apply to all the forms of heart disease.

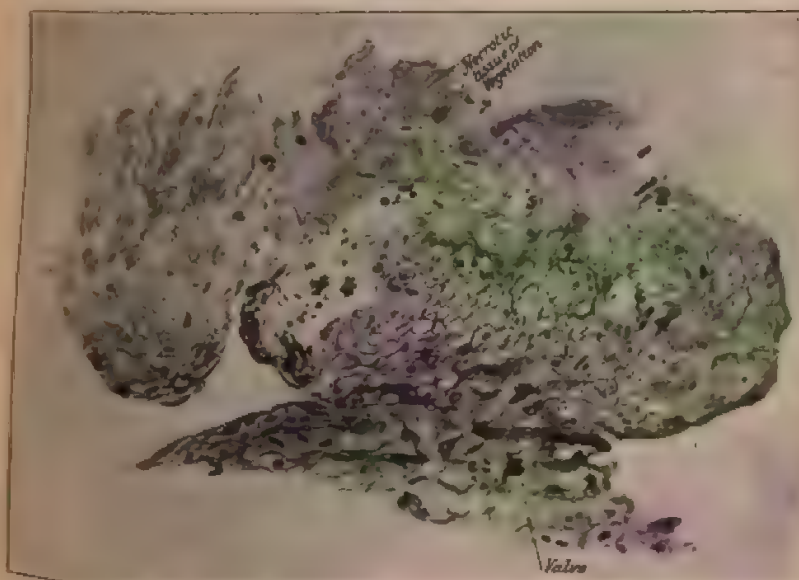
**Pathology.** GENERAL OUTLINE.—Rheumatic fever damages endocardium, myocardium, and pericardium, and to this general injury is given the name *carditis*. While recognizing this tendency to a general damage, it is also clear that in some cases the stress falls upon one structure more than another. Thus the valves are the most frequent



injured, and of these especially the mitral. This is probably because it is the most elaborate, and the best supplied with blood, and I would compare it, for this reason, to a large joint.

The first step in the morbid process is the deposition of the micrococci in the subendothelial layer of the fibrous tissue of the valve or pericardium. Then follow swelling of the connective tissue, dilatation and even rupture of blood capillaries, and exudation. If the process is severe, the connective tissue is destroyed and becomes necrotic, and the lining endothelium which lies over the damaged area of valve or pericardium is also injured. In the mean time the protective processes come into action. The connective-tissue cells multiply, and the leuko-

FIG. 145



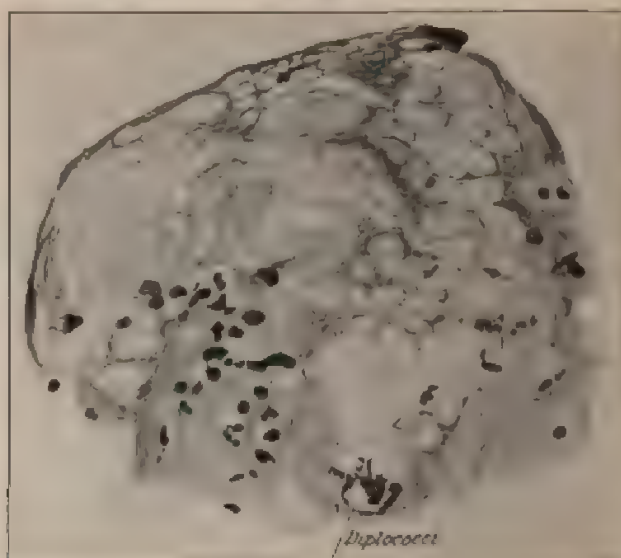
Rheumatic endocarditis, showing granulation in the necrotic stage.

cytes, escaping from the bloodvessels, take up the bacteria. The endothelium, where it is not fatally injured, does the same duty, and, eventually, a balance is usually struck between the disease and the reaction. The bacteria are destroyed, but the tissues, on the other hand, are often irreparably damaged, and need to be patched with scar tissue. This sequence of events, the march of the disease, the march of the resistance, the struggle and imperfect victory, is the history of active rheumatic heart disease as generally met with in childhood.

The cardinal variations from this type are two. One of these is a continuous smouldering inflammation in which the entire thickness of the valve or pericardium is implicated, and the connective tissue throughout them is swollen and infiltrated with leukocytes. This process is

very slow, but, eventually, there is great damage to the connective tissue, and the contraction which results is extreme. It is well exemplified by the true mitral stenosis. The other deviation from the ordinary type is a far more virulent process, in which the balance between the disease and resistance, far from being equal, is greatly in favor of the disease, and the bacteria multiply in the local lesions with great rapidity. It is well exemplified by the rheumatic form of malignant endocarditis. The result is a remarkable one. Large vegetations form upon the valve, and the micro-organisms are scattered by the blood stream in every direction. Here, again, it must be pointed out that many will not accept this interpretation, but maintain that all cases

FIG. 146



Rheumatic endocarditis, showing diplococci.

of malignant endocarditis are the result of mixed infections with septic micro-organisms. (See Plate XX., Figs. 145, 146, 147 and 148.)

**ENDOCARDITIS.** The *cardiac valves* are damaged in this order of frequency: (1) the mitral, (2) the aortic, (3) the tricuspid, and, very rarely, (4) the pulmonary. I am convinced that the statement that rheumatism only affects the left side of the heart, because the blood there is arterial, is one of those ideas which appeals rather to the imagination than to the reason. The mitral and aortic valves are affected simultaneously or in rapid sequence with considerable frequency, but any severe affection of the tricuspid valve is very rare.

The local lesions in the mitral valve take the form of small, pinhead-sized vegetations ranged along the lines of contact of the segments, and they are usually situated upon the auricular surfaces of the mitral an-

PLATE XX.



Rheumatic Endocarditis, showing the Commencement of a Vegetation.

Section through a cusp of the pulmonary valve. (Herman). All the valves were damaged by rheumatism.

- A. The early vegetation formed by broken connective tissue.
- B. Endothelial lining of upper surface of valve.
- C. Connective tissue framework of valve.
- D. Endothelial lining of under surface of valve.

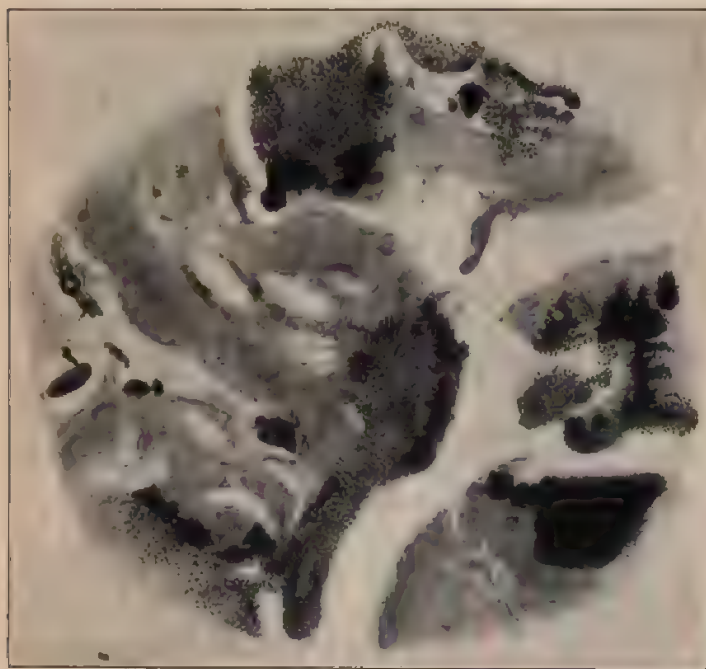
111



tricuspid valves and the ventricular aspect of the aortic valves (see Fig. 149), but in severe cases, especially of the malignant type, they are met with on both aspects, on the chordæ tendinæ, on the inner surface of the walls of the heart itself, and at the base of the aorta (Fig. 153). The various stages in the production of the lesion are seen in Plate XX., and Figs. 145 and 146.

The edge of the valve in the earliest stage is reddened, in the later stages, the vegetations have a waxy yellow appearance, and in some malignant cases these vegetations reach a large size. If the reader will turn to Fig. 145 he will see that in the necrotic tissue there are no micro-

FIG. 147



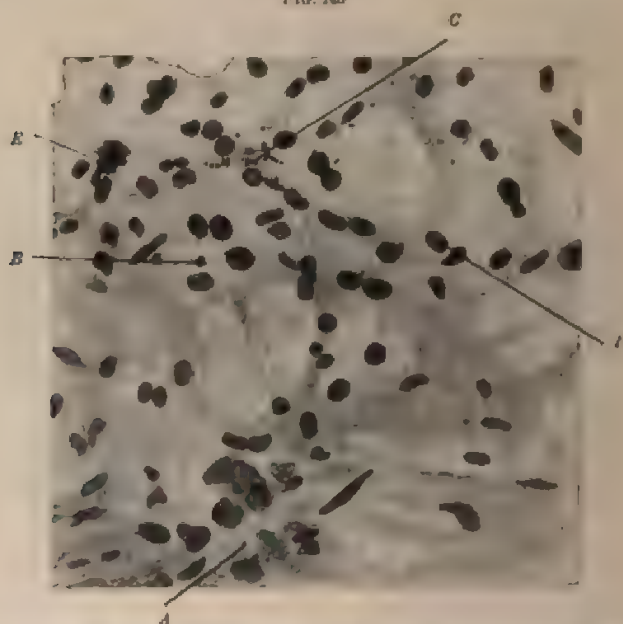
Malignant endocarditis. Rheumatic. Showing masses of diplococci in the necrotic tissue of the vegetation.

organisms, while in Fig. 147 they are numerous. The former is an instance of the usual simple endocarditis and the latter of the malignant type. This is the secret of their difference, and brings home very vividly the great clinical fact that simple acute rheumatic endocarditis is never fatal in the acute phase. Why should it be? The micro-organisms are destroyed, the valve heals, and the acute stage is very often over even when the patient succumbs to pericarditis. Cultures from such valves are generally negative. But it is far otherwise with malignant endocarditis—a veritable bacterial volcano—for that disease is usually fatal (Fig. 147).

When the process of healing is studied, two types of great practical importance can be recognized. The first is that in which the free edge of the valve is contracted and crumpled by scarring, and its edge thick and uneven; it is the result of the bursting out of the vegetations along the edge of the valve. The result of such a lesion is *incompetence*.

The other is represented by a welding together of the segments of the valve, a shortening of the chordæ, and a general thickening of the valvular ring. It is the outcome of a chronic smouldering inflammation which affects the entire thickness rather than the margin of the valve. The result is a *stenosis* in which the opening, which may only admit the top of a pencil, may be slit-like, or may keep its circular outline, and then resemble the orifice at the base of a funnel.

FIG. 143



Rheumatic pericarditis. Section through visceral layer, showing the diplococci. A, cardiac muscle; B, visceral pericardium; C, diplococcus; D, connective tissue cells; E, inflammatory cell.

There are connecting links between these two great types of healing, but in their pure forms they represent the two different processes alluded to above. The aortic valves are seldom very greatly damaged, and in most cases a slight thickening and crumpling are the result of the inflammation. The tricuspid valve is infected more frequently than is generally known, but usually only to a slight and practically insignificant extent, yet there are occasional examples in which it is greatly damaged, and there may in these cases result in later life a combined mitral and tricuspid stenosis.

The pulmonary valve is damaged so rarely and so slightly as to need no further comment.

**THE PERICARDIUM.**—Pericarditis is the result of the more severe types of rheumatic fever, and may be a cause of death.

In very acute cases the pericardium is reddened, and there is a moderate amount of exuded fluid in the sac which is turbid or even blood-stained. In other cases, of longer duration, there is much fibrinocellular exudation (Fig. 152) which adheres to both layers of the pericardium. With this there are also flakes of exudation lying free in the cavity, and the fluid is more opaque.

In still other cases there has been an attempt at recovery and the two layers are found adherent with recent plastic exudation, and, finally, the evidence of an old pericarditis may be discovered by the

FIG. 149



Simple rheumatic mitral endocarditis. To show the line of vegetation upon the auricular surface of the mitral valve.

Occurrence of partial or total adhesion of the two layers by connective-tissue formation.

The actual morbid processes in pericarditis start in the subendothelial layers of the pericardium, and it is even then general, essentially the result of numerous individual foci of inflammation originated by the infective agent.

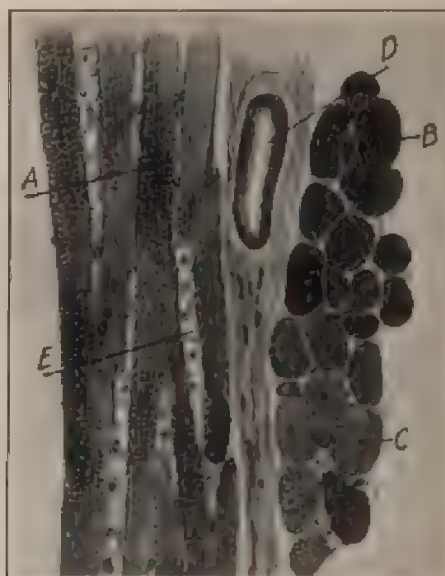
The yellow liquid pus seen in suppurative pericarditis is not found in true rheumatic cases.

If the pericardium is found to be adherent it is important to recognize the extent to which this has occurred. Is it simply an adhesion of the

two layers, or in addition are there extensive pleuropericarditis and mediastinitis chaining the heart to the chest wall and to the lungs? In such cases not only the internal endothelial surface of the parietal pericardium has been much injured, but the inflammation has spread to the cellular tissues external to the pericardium. Finally, a large pericardial exudation is rare in rheumatic pericarditis, and it is from the exudation when it is fibrinoplastic that the diplococcus can be most easily isolated.

THE MYOCARDIUM is frequently damaged, but since the cardiac wall consists in great part of very special tissues—the muscles and nerves—in order to grasp the true meaning of the changes the word myocarditis

FIG. 150



Rheumatic carditis. Fatty change in cardiac muscle. A, fatty granules. B, fatty granules in horizontal section; C, hyaline change. D, bloodvessel.

is better set aside. In place of this, two processes will be considered: the first, which damages the muscle, a subtle bacterial poison; the second, that which sets up inflammation in the region of the bloodvessels and supporting connective tissue.

It would be wrong to attribute such a change as the fatty degeneration of the muscle entirely to a slow disturbance of nutrition resulting from damage to the bloodvessels, and thus make it dependent upon the inflammatory changes; for intravenous inoculation of a monkey with the diplococcus has produced these fatty changes within as short a time as four days.

The morbid processes in the muscle, which are of the greatest practical



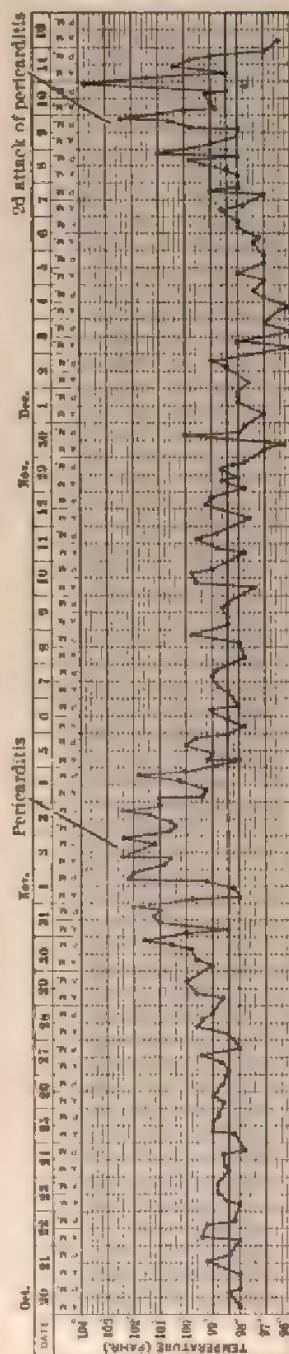
moment, have not yet been thoroughly worked out, for we do not yet know what governs their frequency or severity, though we thoroughly realize their existence. There is no doubt that they are far more extensive in some cases than in others, and as the outcome of an examination of some 40 cases of rheumatic carditis, it seems to me that severe injury is especially found in cases of virulent pericarditis. But this injury does not spread inward from the pericardium, for such lesions are to be found distributed in patches in the neighborhood of the small bloodvessels throughout the wall of the ventricles (Fig. 150), and what is still more convincing may be found without any pericarditis. It is only to be expected that the morbid changes will be most evident immediately under the inflamed pericardium, for the diplococcus is deposited in the subendothelial tissues, but that the injury spreads from the valvular ring or pericardium, as a drop of ink spreads on blotting paper, is not correct.

The most definite alterations in the muscle are the fatty ones as shown in Fig. 150. Nuclear changes, fragmentation of the fibres, diminished and exaggerated striation have also been observed. The minute bloodvessels in the supporting connective tissue are sometimes ruptured and minute extravasations result. There is also perivascular exudation, and in some chronic cases there are found interstitial and perivascular fibrosis. The papillary muscles are sometimes very much injured by the poisons, especially when there is endocarditis; their function is thereby impaired.

In cases of heart disease with marked hypertrophy the muscular fibres are not only more numerous, but larger than normal.

**Symptomatology. Early Warnings.**—Upon these early warnings I would lay the greatest stress, for, so far as one can

FIG. 151



Temperature chart from a case of virulent and fatal rheumatism, illustrating relapsing pericarditis.

see, there is no possible cure for severe organic heart disease and the great hope lies in its prevention.

In all cases of rheumatic fever, however slight or doubtful, it must be surmised that the heart is damaged until it is certain from careful examination that it has escaped. By rheumatic fever I do not mean merely articular rheumatism, but all the varied manifestations of the disease, such as chorea, myalgia, erythema multiforme, nodule formation, bronchopneumonia and pleurisy, arthritis and tonsillitis. Any of these manifestations are warnings, and the more important because the early heart disease of childhood is notoriously destitute of striking clinical symptoms. I have in mind a child whose mother remarked to me that he ran about "almost like other children," yet to my knowledge he had a pericardium adherent from a previous and severe pericarditis, and disease of both the aortic and mitral valves. This is an experience common to all who are acquainted with cardiac disease in childhood.

There are still earlier warnings than these recognized manifestations, some of which must be equivocal, yet in a child of rheumatic stock they should arouse suspicion. Wasting, slight fever, vague pains, especially in the epigastrium, nervousness and night terrors, anemia and epistaxis are among them, and though no one would pretend to be confident that rheumatic fever was their explanation, experience has shown that such vague symptoms may terminate in undoubted cardiac rheumatism.

It is the many manifestations and insidious course of rheumatic fever in the young which distracts the attention from the heart and leads to lamentable oversights. On the other hand, the heart may be the first organ severely attacked, and there are numerous cases in which the symptoms compel our attention to it. Thus the child complains of pain over the heart and shortness of breath, or the mother may notice the thumping excited impulse.

**ACUTE DILATATION OF THE HEART.**—From what has been already written concerning the morbid anatomy, it will be readily understood that severe rheumatic infections damage all parts of the heart and cause a carditis, but many cases, whether acute or subacute, need not necessarily be severe, and, even if they are severe, the attack must have a beginning, and that beginning usually manifests itself as *acute dilatation of the heart*. From this there may be complete recovery. If these patients could always be brought under medical observation at this stage it seems probable they might be saved from many dangers. Unfortunately, this often does not occur; moreover, in some cases the dilatation is overlooked.

The *symptoms* observed in dilatation are as follows: There is a slight rise of temperature (99.5° F.). The child may be a little short of breath and pale. The rate of the pulse may be increased to 90 or 100, and be irregular in rhythm and low in tension. The cardiac impulse is diffuse and the area of deep cardiac dullness increased to the left. The first sound in the region of the impulse is short, and at the base the second sound over the pulmonary area accentuated.

There may also be a soft, systolic, whiffing murmur, heard most distinctly internal to the nipple line.

It is a valuable education, although it needs some expenditure of time, to ascertain the limits of the deep cardiac dulness, and to mark them upon the chest wall with an aniline pencil. In this outline three landmarks should be also indicated, the left nipple, the midsternal line, and the subcostal angle; the chart can then be traced from the chest wall upon surveyors' paper and kept for future reference.

Experience has amply shown that this stage of dilatation may often be demonstrated before any severe cardiac lesion has occurred and that the latter may subsequently follow. Whether in any particular case its detection and treatment may have assisted in warding off the graver lesions, it is naturally impossible to assert, but there is good reason, in view of the value of rest in rheumatic heart disease, to hope that this has happened.

There is a certain danger of overcaution, I admit; but I feel so strongly upon the value of this early sign of disease that if this article should help to impress the importance of acute dilatation upon the minds of those who are not alive to its value, it will have done some service to children.

This condition of dilatation occurs in all cases of rheumatic carditis; it complicates the valvular lesions and pericarditis, and is the great cause of cardiac failure in a heart already damaged by former attacks.

When the dilatation is more severe, all the signs mentioned above will be emphasized, and, further, the systolic murmur can then be traced out ward beyond the left nipple. Yet even from severe dilatation there may be complete recovery.

How is it known that with this acute dilatation there is not mitral endocarditis? In man and in animals there are examples on record of death from acute rheumatic dilatation without endocarditis. If, then, dilatation can reach this pitch without endocarditis, it is legitimate to argue, in view of the complete recovery, that the less severe condition may also occur and with greater frequency. Even if there should be a slight degree of endocarditis, that, in itself, would not account for the dilatation.

**ENDOCARDITIS.**—Almost insensibly upon this early dilatation there may follow definite endocarditis, while in many cases, doubtless, the two processes occur simultaneously.

The *symptoms* are quite unobtrusive, a little palpitation, some vague pains over the chest and epigastrium, pallor and a little fever are the usual ones. The temperature may run up a degree or two. Attentive observation of the character of the first sound at the impulse and of the second sound at the aortic area will be needed. In *mitral endocarditis* the first sound becomes short and ill-defined, and then a soft systolic murmur appears which will replace it to a greater or less degree. Auscultation should be practised with the child in the recumbent position as well as sitting up, and both external and internal to the left nipple line. The bruit, at first perhaps only to be heard occasionally,

was reduplication of the second sound of the heart. The 'luf-tüt-tüt' perhaps gives the impression that is conveyed. This is an important physical sign, and implies that the aortic valve is thickened, and has been the seat of actual inflammation.

The history of early *aortic endocarditis* is very similar to that of *mitral endocarditis*, but needs even closer observation.

The aortic second sound becomes faint and for a day or two is inaudible; then there appears a faint systolic bruit, and, lastly, a faint systolic murmur. This latter murmur is frequently heard better to the left of the sternum, or behind the sternum, or even in the tricuspid area. The aortic cartilage.

Within a fortnight the collapsing pulse of aortic regurgitation may be quite definite, and already the radial artery may be diminished in calibre.

The *tricuspid* valve does not often show signs of injury, such is the case the same sequence of events will be noticed in the case of the mitral valve.

Thus, in this quiet and insidious way is a life ruined by heart disease, and the vital importance of the early dilatation of the heart home to us.

PERICARDITIS.—It is in the severe cases of rheumatic fever that pericarditis occurs in a first attack, and, moreover, it is the most common complication. But, in spite of this, I would warn the practitioner of the danger of supposing that a heart is not greatly damaged because there is no pericarditis or, on the other hand, of thinking that because there has been pericarditis the damage is irreparable. There are many cases in which there is endocarditis with disease of the pericardium, and in which the action of the heart is very excited, and in which the action of the heart is very excited, and in which the action of the heart is very excited. So far as a good recovery is concerned, the prognosis is ill-omened. On the other hand, a fleeting pericarditis may be the heart but very little the worse for the attack.

With the onset of pericarditis these definite symptoms are noticed. The temperature rises to 100° or 101° F. and



*friction rub*. It is a physical sign of the utmost value, and the most careful study at the bedside is needed to recognize its different characters.

Pericardial friction is usually heard, at one time or another, in the course of rheumatic pericarditis, and, on this account, rheumatic pericarditis is an easier condition to diagnose than suppurative pericarditis, in which a rub is usually never heard at all. Most commonly pericardial friction is a to-and-fro rubbing sound which to the trained ear is evidently quite superficial; pressure will modify it, but the tenderness over the cardiac region should make the attempt a cautious one.

Often at first quite soft in character, later it may be loud and harsh and obscure all other auscultatory signs. Sometimes it is only heard during systole and then, if it is faint, it may be mistaken for an endocardial bruit. There are writers who hold that the to-and-fro friction rub cannot be mistaken for an endocardial sound, but the difficulty may be a very real one when a double aortic murmur is present as well as pericarditis. Generally the first spot at which the rub is heard is over the large vessels at the base of the heart, and another favorite area is at the horizontal nipple-level immediately to the left of the sternum. This area in either case may increase with great rapidity, and within twenty-four hours the friction may become general.

Because of its extreme value, I place this physical sign before all the other evidences of pericarditis, but it is necessary also to make a careful and complete examination of the heart on the classical lines of inspection, palpation, percussion, and auscultation. It will be found that, in the severe and acute cases, the action of the heart is greatly excited and the impulse diffuse. The area of deep cardiac dullness is increased and may, as the illness advances, become literally enormous. It is an increase upward as well as laterally, and with it there is also an increase in the area of superficial cardiac dullness.

The stethoscope, in addition to demonstrating the pericardial friction, conveys better than any other means the reality of the cardiac excitement, and by it in most cases a mitral systolic bruit can be detected at a deeper level than the friction rub, for with pericarditis there is as a rule endocarditis.

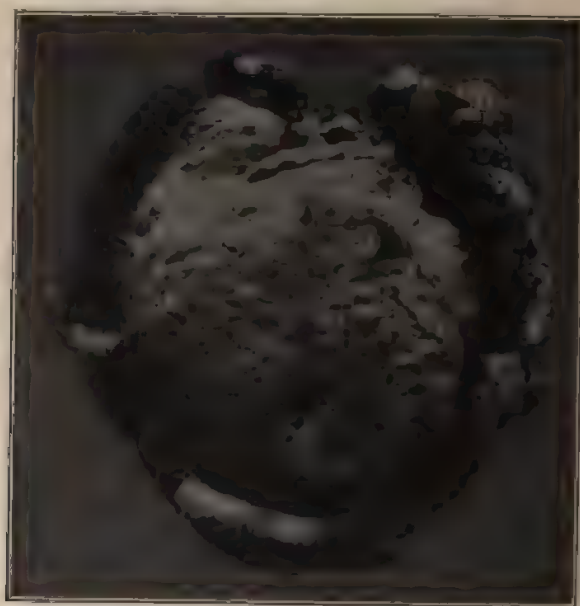
The excited action of the heart, the rapid sounds, the bruit and friction rub, together, give a curious tumultuous noise which baffles description, but which, when once heard, is very characteristic of rheumatic pericarditis.

All are agreed that there is an increase in the quantity of fluid in the pericardial sac coincident with the early inflammation, and all are agreed that there is comparatively often in the later stages of pericarditis a very great increase in the area of precordial dullness. The usual explanation formerly given for this great increase in the cardiac dullness was that there was much exudation, but now we know it is mainly the *dilatation* of the heart and not the *fluid* which explains this occurrence.

At first, then, some muffling of the cardiac sounds may be detected, and the early friction may become faint or even disappear, and these

mean that some considerable exudation has taken place. Yet I believe myself correct in stating that far more frequently this phase is not to be detected at all, and yet the area of pericardial dulness increases both to the right and to the left and upward. A large effusion in the rheumatic pericarditis of childhood must be extremely rare. I have never seen one, after death, so large that it needed paracentesis during life, but I have seen many which during life were thought to need it, and in a few of these the thought determined action, a needle was introduced, and blood drawn from the heart. There is then a different explanation needed for the phenomenon of the enlargement of the precordial dulness, and, as has been already insisted, that explanation is *acute dilatation of the heart itself*.

FIG. 152



Rheumatic pericarditis. The pericardium has been opened and shows the plastic exudation.

Acute general pericarditis is clearly a very dangerous condition, not so much because of the immediate risk to life, but because it implies in most cases a *carditis*, which leaves the heart permanently weakened. It is difficult to give precise dates for the duration of the acute stage, cases differ so in this respect. One child may have definite pericarditis and yet all the physical signs clear up in a week; another may drift into a subacute condition which lasts for many weeks, while in others again three weeks may be sufficient—not for the heart to recover—but for the signs of pericarditis to entirely subside.

It is so also with the symptoms: some children, except for breathlessness, some pain and fever, suffer but little, and take their food throughout

the attack with enjoyment. But the virulent cases show very plainly the fatal injury to the heart. Thus, livid pallor and rapidly progressing anemia, breathlessness amounting to orthopnea, and pain, are significant symptoms. Even more dangerous ones are continual restlessness, sleeplessness, and vomiting. It is in such cases as these that the precordial area becomes enormously increased, the pulse rises to 130 to 150, and is small, irregular, and of low tension. Yet there is no striking edema, but toward the end of life there is some puffiness of the ankles or lower extremities. With the failure of the heart, the liver enlarges

FIG. 153



**Malignant endocarditis.** The heart of a child; the aortic valve is exposed and shows a large vegetation, the result of malignant endocarditis.

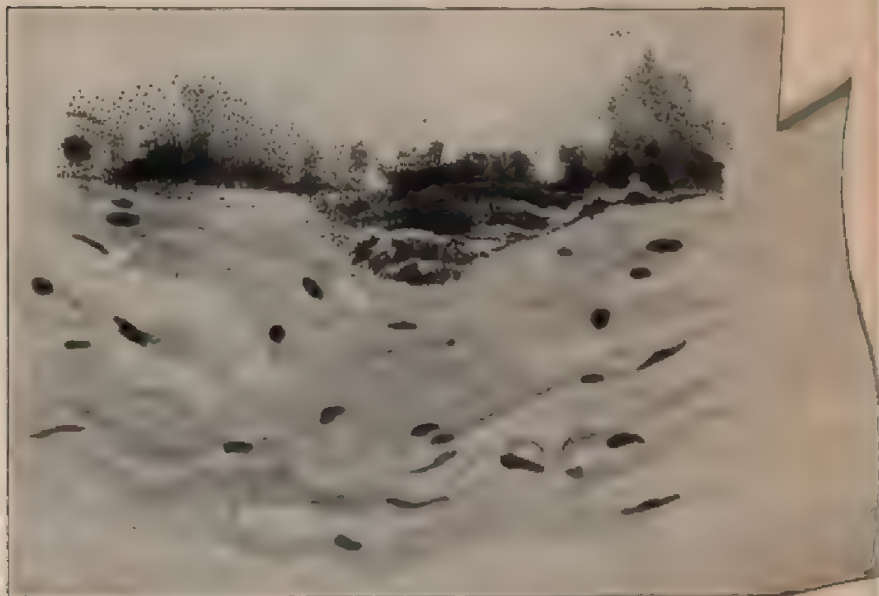
and may extend below the umbilicus and be tender to the touch. The lungs become congested at their bases, and fluid may be found in both pleura. Very often also there are true rheumatic pleurisy and pleuro-pericarditis, and in rare cases an acute edema of the lungs develops with great rapidity and causes a rapid death. The urine in these later stages is scanty and albuminous.

When death occurs in a first attack of carditis—a rare event—these are the symptoms to be expected, and the actual cause of death is generally sudden cardiac failure. The temperature for days may be subnormal. Fortunately the more usual course of pericarditis is toward



recovery, and then the general condition improves, the face looks less pinched and is a better color, the pulse and respiration rate diminish, the area of cardiac dullness lessens, and the temperature quietly approaches the normal. The liver becomes smaller and the urine free from albumin, if any has been present. The recovery, it is true, may be slow and interrupted by relapses, but it is far more common than the fatal result. Yet it must be admitted that this recovery is not, as a rule, a perfect one, for the opposing endothelial surfaces of the pericardium have been damaged, and adhesion, more or less complete, is to be expected.

FIG. 154



Endocarditis, chorea, and rheumatic fever. Numerous diplococci are present in the valve tissue. The vegetations are small as in simple rheumatic endocarditis, but there are numerous diplococci.

Lastly, it should be clearly recognized that at the bedside rheumatic pericarditis must often be looked upon as only one manifestation of the rheumatic infection, and that the true history of the illness is one in which this pericarditis is only an incident. The temperature chart (Fig. 151) shown on page 703 is a good illustration of this truth. (See Figs. 149, 152 and 154.)

**MYOCARDIAL DAMAGE.**—This can be divided roughly into two groups: the first a small one in which are placed the rare, acute, and fatal cases of rheumatic myocardial disease, which will be treated of later; the second a larger one, difficult to recognize with confidence, but probably more common than is usually supposed. In these the damage is less severe, but the cardiac valves and pericardium escape, or, at the most, the mitral valve is slightly damaged.



ases in this second group begin, just as other rheumatic cases of erate severity, with some dilatation of the heart and perhaps other s of rheumatism, but the heart remains large and the pulse rapid irregular; the child is breathless, anemic and excitable, and very y tired. There may be a systolic murmur which disappears with very. It is difficult to be sure there is not some endocarditis, but if e is it cannot be responsible for the cardiac weakness, which is e out of proportion to the amount of endocarditis present, and some- s very persistent. But before the conclusion is arrived at, that the iac wall is at fault, it is important to assure one's self that there has been a previous pericarditis, or that the mitral valve is not narrowed ome insidious and intractable endocarditis.

## CHAPTER XXIX.

### CHRONIC RHEUMATIC HEART DISEASE—TREATMENT OF RHEUMATIC HEART DISEASE.

#### CHRONIC RHEUMATIC HEART DISEASE.

##### *A. The Stage of Compensation.*

**Symptomatology.**—When the acute rheumatic illness is over, the damage which is generally left behind is slowly corrected by the development of hypertrophy of the heart. In this way health and strength are restored, and if not to the former degree of perfection, yet often so far as to enable the child to live a happy and useful life. The lesions are now said to be *compensated*, and it is all important to recognize the factors in this compensation.

**Mitral Regurgitation.**—During the systole of the left ventricle some blood is forced back through the incompetent mitral valve into the left auricle. The left auricle must then be dilated, for it will contain at the end of its diastole the usual supply of blood from the pulmonary veins, together with the amount regurgitated. The left ventricle, also, will be dilated to receive a larger supply on the systole of the auricle. Both chambers will hypertrophy in order to properly discharge the increased quantity of blood. The musculature of the left auricle is, however, but comparatively feeble, and thus it follows that, when the regurgitation is considerable, difficulty will be felt in the pulmonary circulation. In order to overcome this, the right ventricle is called upon for increased effort and so hypertrophies. When this hypertrophy begins to fail, the tricuspid ring dilates with the general dilatation of the ventricle, and relative incompetence of this valve will result. This incompetence, in turn, is to some extent compensated for by dilatation and hypertrophy of the right auricle. The power of this auricle is but slight, and so, last of all, the systemic veins feel the strain of the back pressure, and the cardinal signs of tricuspid regurgitation are manifested.

**Mitral Stenosis.** In pure mitral stenosis, the difficulty is a more serious one, for immediately in front of the comparatively weak left auricle there lies the narrow opening of the mitral valve. Hypertrophy of the auricle is needed, and soon the strain is felt also in the pulmonary circuit. The right ventricle must come to the rescue, and hypertrophy. When the right ventricle fails, the sequence of events is as in mitral regurgitation. A strong right ventricle is the safeguard of the patient. The left ventricle in severe cases receives less blood than normal and the muscle may atrophy. The small output of this ventricle is shown by the stunting of the child in development.

**Aortic Regurgitation.**—The result of this lesion is that during diastole a certain quantity of blood passes back through the damaged valve into the left ventricle, which contains in addition the usual supply from the left auricle. The left ventricle is dilated to receive the increased quantity, and, to carry out its increased work, it hypertrophies. When aortic regurgitation is very considerable and the dilatation of the left ventricle is great from failure to cope with the strain, the mitral valve may become relatively incompetent and mitral regurgitation will then supervene upon aortic regurgitation. The enormous enlargement of the left ventricle that can result in the adult from aortic regurgitation is not often seen in the child, for aortic lesions are rarely severe in the young.

**Aortic Stenosis.**—The strain is first felt during the systole of the left ventricle, and in order to force the blood through the narrowed orifice there must be hypertrophy of the left ventricle. Later, when the ventricle fails, there will be dilatation and perhaps mitral incompetence, followed by the secondary results of mitral regurgitation.

**Tricuspid regurgitation and stenosis** can be understood from what has already been written upon the similar conditions at the mitral orifice.

**Combined Lesions.**—A very common result of rheumatic fever is a combination of *mitral stenosis and regurgitation*; the strain then is upon the left auricle, pulmonary circuit, and right ventricle.

Another result is mitral and aortic regurgitation, in which case much hypertrophy of the left ventricle is needed.

When there is a *generally adherent pericardium*, there is, as a rule, some general dilatation and hypertrophy of the heart, but it is difficult to recognize to what extent this enlargement of the heart is the result of the adhesion, of the usually coincident valvular disease, or of the myocardial weakness.

There are exceptional cases in which the heart, without the occurrence of endocarditis, is strangled and atrophied by extreme pericardial adhesion and thickening.

**Diagnostic Points.**—The clinical features of these cardinal lesions are as follows:

**Compensated Mitral Regurgitation.**—There is often nothing characteristic in the aspect of the child, though there may be some breathlessness on exertion and cough, a tendency to bronchitis, and a slightly purple tinge to the lips and face.

The pulse is more rapid than usual, easily compressible, of fair volume, and either regular or very slightly unequal in the strength of the individual waves.

The cardiac impulse is forcible, and situated external to the left nipple line in the fifth space or touching the fifth rib, and the cardiac area is increased to some extent both to the right and left. At the impulse there is a systolic murmur traceable toward the axilla and often audible at the back on the left side in the infrascapular region. The second sound at the pulmonary area is accentuated and reduplicated.

If, as is so often the case, there is some slight mitral constriction as

well, there will be heard, in addition, at the impulse either a slight rumble immediately before the first sound, or the same in mid-diastole, or immediately following the second sound. This rumbling sound is, as a rule, strictly localized to the impulse and not conducted. On palpation the hand may feel a presystolic thrill.

*Aortic Regurgitation.*—If this is well marked, there are usually pallor, nervousness, and some dyspnea upon exertion; but none of these symptoms may be present. The pulse is increased in rate, it is usually regular, and the wave large, sudden, and ill-sustained. The radial artery may pulsate visibly, and the pulse may be audible on putting the wrist to the ear. There is capillary pulsation on pressing the "quick" of the nails, or stroking the forehead smartly. The impulse is forcible, and the area increased to the left and downward. The cardinal sign is a diastolic murmur, sometimes long and blowing, at other times short and soft. The position of maximum intensity is very variable; it is often heard most distinctly in the third *left* space close to the sternum, sometimes at the inner end of the second right space, or behind the sternum at that level, or over the ensiform cartilage. It can, in some cases, be traced down the right margin of the sternum, or even be heard at the impulse. This murmur, in my experience, is more localized in the child than in the adult.

*Aortic Stenosis.*—This is a very rare condition in children. There is breathlessness on exertion, and the growth of the child is stunted. The pulse is rapid, the wave small and not easily compressible. The impulse is forcible and displaced downward and outward. The area is increased, as in aortic regurgitation. The cardinal sign is a harsh systolic murmur which gives a systolic thrill to the hand. It is heard most distinctly over the aortic cartilage, and is traceable into the large vessels of the neck.

*Tricuspid Regurgitation.*—This is so intimately connected with the details of failing compensation that it will be dealt with under that heading.

*Adherent Pericardium.*—I think it is impossible to diagnose this in a child with any certainty, except in rare cases. When, however, there is indurative mediastino-pericarditis, which will be described later, there may be sufficient evidence. I have seen experienced observers time after time make the diagnosis of adherent pericardium upon the evidence that is usually accepted as sufficient, and yet be wrong. Adhesion is a very common result of pericarditis, and it is a common event in the autopsy-room; its occurrence during life can thus be often guessed correctly from the history of the illness, but it is no more than a likely guess.

When there is fixation of the heart to the chest wall and pleura, then the following are the more important physical signs of adhesion:

- a. Immobility of the apex beat, upon deep respiration and upon change of position. This sign is of little value in children, for a large heart, without pericardial adhesion, will not move with a change of position in a small chest.



There is systolic recession of the intercostal spaces and cartilages to the left of the sternum. In some cases the epigastrium and lower end of the sternum are drawn in with systole, and, as Dr. John Broadbent has pointed out, the sides and posterior walls of the lower part of the thorax may also show this same retraction.

A diastolic shock may be felt by the hand, placed over the area of retraction to the left of the sternum, and is due to the elastic recoil of the chest wall at the commencement of diastole. There will also be cardiac enlargement, due to hypertrophy and dilatation, and the respiratory movements of the diaphragm may be embarrassed by firm pericardial adhesions.

Diastolic collapse of the veins, in conjunction with systolic recession of the intercostal spaces, as described by Friedreich, does not seem to have met with general acceptance.

Lastly, the physician may find that the damage to the heart is greater than would be expected from some simple valvular lesion, and in this way be led to suspect pericardial adhesion.

*Multiple Valvular Lesions.*—It is not uncommon for two valves to be damaged simultaneously in an attack of rheumatism, and so far as children are concerned, where aortic and mitral incompetence are found, it is more probable that both arise from the rheumatic infection than that the mitral regurgitation is a secondary result of the aortic regurgitation. In other words, the mitral regurgitation in these cases is due to endocarditis and not to relative incompetence.

The aortic and mitral lesion is the most usual combination and it is a serious condition. In four consecutive cases, coming under my notice, three died within eighteen months from rheumatic complications, and the fourth is anemic, highly nervous, and short of breath.<sup>1</sup>

The evidence of the double lesion is usually definite, but unless the practitioner is careful he may overlook the aortic disease, the diastolic murmur of which is often most clearly heard to the left of the sternum.

*Mitral Stenosis.*—It might seem that this, of all the lesions, was the most stationary, yet it is not so, and close inquiry will prove that it is often steadily progressive. Advanced mitral stenosis is rare under twelve years of age, and yet mitral endocarditis is very common, and may be met with as early as four years or younger; certainly after six years it is common enough, and often severe. If, then, mitral stenosis was the usual result of the healing of an inflamed valve, it should be common enough, for between six and twelve years of age there is ample time for the processes of scarring and cicatricial contraction.

Then, again, the cases of mitral stenosis which are met with have usually one remarkable feature in their illness, and that is the absence of any very definite history of an acute attack of rheumatism, although close inquiry will often elicit a prolonged history of indefinite rheumatism. There can be little doubt, I think, that this form is the result of a persistent, smouldering, rheumatic inflammation of the entire

<sup>1</sup> Since writing this article I have published in the British Medical Journal, October 7, 1905, details of twenty-one such cases.

thickness of the mitral valve, mitral ring, and chordæ tendinæ, which I would compare to chronic periarticular rheumatism. That the inflammation was a peculiar one was the opinion of Dr. Sanson some twenty years ago. To illustrate my meaning by analogy, it is comparable to the fibroid type of tuberculosis of the lungs.

Although there may be no definite history of rheumatism at all, in a certain number of cases chorea, of a persistent and intractable type, is a witness to the activity of the rheumatic process.

Mitral stenosis is well known to be more common in females, and I believe this to be because all rheumatic affections are more liable to be chronic and smouldering in the female.

The commencement of mitral stenosis is most insidious and often enough the child is never brought to the doctor until the disease is well advanced, for there may be no pain nor discomfort. When its development can be traced these are the most usual phenomena:

The pulse is increased in rate, at first regular, small and not very easily compressible. Sometimes it feels like a thin wire.

If there is a regurgitant bruit this slowly disappears and leaves a first sound which is short and ends abruptly. Then the curious reduplication of the second sound (tut-tut), which points to some thickening of the valve becomes more pronounced and longer, and may occupy most of the diastole. With this development, a presystolic thrill can be felt in the region of the impulse, and the pulmonary second sound is accentuated. Finally, the so-called reduplication at the impulse becomes a well-marked presystolic murmur, leading up to a short, sharp, first sound.

The clinical picture of severe mitral stenosis is an interesting one.

The small output of blood from the left ventricle in severe mitral stenosis leads to stunting of the growth of the child. There is often a persistent, red flush on the cheeks, and the eyes have a very curious translucent brightness.

The disposition is singularly patient and attractive, a result partly of the refining influence of invalidism and partly of the altered cerebral circulation due to the valvular defect.

The circulation in the extremities is poor and the fingers often blue and cold. This imperfection in the circulation may reach such a degree that, when the heart fails, gangrene of the extremities may result.

#### *B. Ruptured Compensation.*

The compensation of a damaged heart is upset by many influences, but more especially by a relapse of rheumatism. Overexertion, rapid growth with anæmia, pulmonary affections, nervous strain, and infectious diseases are all of them occasional factors. Again, when compensation has been effected only with great difficulty, and its margin of reserve is consequently very narrow, even the ordinary exertion of every-day life may be too much for the crippled heart.

The breakdown in the health of the child is thus, as a rule, a complex

process, in which active rheumatism, and failure of the heart to perform its function on account of valvular defects, take varying prominence. In some cases it is the active rheumatism that is the prominent factor; in others it is the failure of compensation.

The group in which active rheumatism is prominent will need no further description, for to understand them we have only to apply the principles, which have been already given under acute rheumatic heart disease, to a heart already maimed by previous attacks.

The second group includes those cases in which the rheumatism has merely stepped in and pushed the heart, as it were, over the brink of the precipice, upon which it was already standing. These will need further description.

*Dilatation and Hypertrophy of the Heart.*—It will possibly make the subject clearer if a few lines are devoted to dilatation and hypertrophy apart from the valvular defects.

Dilatation may either be a necessary result of valvular incompetence—a provision for the accommodation of blood which has leaked through the opening—or a result of the failure of the myocardium to cope with its difficulties.

*Dilatation.*—Where there is dilatation it shows itself by a quickened low-tension pulse which is sometimes irregular. The impulse of the heart is diffuse and tapping or may not be palpable. The area of cardiac dulness is increased to the left, and, if the dilatation is general, to the right as well. The first sound at the apex is short and clear, and is sometimes followed by a soft systolic murmur. When the dilatation is very great, the systolic interval between the sounds is shortened. The pulmonary second sound is usually accentuated. The symptoms are breathlessness, pallor, insomnia, and night terrors, cough and sometimes slight edema.

*Hypertrophy* is salutary, but the very fact of its occurrence is an evidence that the damage which the heart has sustained is a very real one. In the complicated lesions of rheumatic heart disease it is very necessary to search for and recognize the existence both of dilatation and of hypertrophy, which in simpler lesions, such as those of renal heart disease, are so prominent that they can hardly be overlooked.

Hypertrophy is measured by the well-sustained character of the pulse, the forcible and localized impulse, and the enlargement of the cardiac area downward and to the left. The first sound is muffled and slightly prolonged, if the hypertrophy is great.

*Symptoms.*—The early symptoms of ruptured compensation are dyspnea on exertion, cough, precordial pain, palpitation, wasting, epistaxis, loss of appetite, and insomnia. Edema is not a prominent symptom, and is often absent. The cases in which great edema occurs are, as a rule, those in which there is an unusual amount of valvular damage, but a strong myocardium. In mitral stenosis, as Sir William Broadbent has emphasized, ascites may occur without edema.

*Aortic Disease.*—Turning to the heart itself, there are two chief forms of valvular disease to be considered, namely, the *aortic* and



*mitral*. Such cases of aortic disease as I have seen in childhood have, when compensation has failed, either died from acute rheumatic carditis or have lost, for the time being, their aortic characteristics and become to all intents and purposes, mitral in type. I have never met with the sudden syncope which is comparatively frequent in the aortic disease of adult life.

*Mitral Disease*.—When the heart with mitral lesions fails, it is because the right ventricle gives way and tricuspid regurgitation supervenes.

It will be, then, a sufficiently accurate impression if this phase of ruptured compensation be looked upon as essentially a condition of *tricuspid regurgitation*, the features of which it is most essential to recognize. They are:

1. Dyspnea and cyanosis. 2. An enlargement of the heart, especially to the right of the sternum, due to dilatation of the right auricle. 3. The development of a soft systolic murmur in the tricuspid area. 4. An enlarged and tender liver. 5. Full and sometimes pulsating veins in the neck. 6. Congestion of the bases of the lungs. 7. Edema, which is but rarely extensive. 8. A scanty and albuminous urine.

When stenosis of the mitral valve is extreme the pulmonary engorgement is intense, and there may be pulmonary hemorrhage with or without infarction. Further, there may be paroxysmal attacks of palpitation and pain. Even sudden death may occur in mitral stenosis.

For successful treatment, a clear mental picture must also be obtained of the results of the tricuspid regurgitation. The backworking engorgement passes the functions of all the viscera. The liver becomes fatty and nutmeg in appearance; the kidneys are congested and hard; the spleen is shrunken and firm; the stomach is dilated, the walls thickened, and the mucous membrane coated with thick mucus.

Congestion of the bronchial mucous membrane disposes to bronchitis and congestion of the lungs to hypostatic pneumonia. Lastly, the cerebral circulation is disordered, and night terrors, dreams, and insomnia result.

**Physical Signs. Aortic Lesions.**—As I have already mentioned, when compensation fails, the *aortic lesion* is masked. The collapsing character of the pulse and the large wave are modified. The mitral diastolic murmur may disappear completely and leave only a suspicious feebleness or absence of the second sound in the aortic area.

This masking of the lesion is of considerable importance, and the cautious physician will not give a definite opinion upon the exact condition of the heart when he has seen the patient but once, and the patient is in this stage of failure. For, when the heart rallies the aortic murmur will reappear, to the surprise of a hasty diagnostician, and an unsuspected aortic lesion will become apparent.

**Mitral Lesions.**—The heart with mitral lesions is variously affected by ruptured compensation.

In *mitral regurgitation* the pulse becomes rapid and, sometimes exceedingly irregular. The systolic mitral bruit is more prolonged



and may entirely replace the first sound. The previously, forcible impulse of the left ventricle becomes diffuse and tapping, and the accentuation of the pulmonary second sound disappears.

In *mitral stenosis* the pulse becomes irregular and more easily compressible. The presystolic murmur and thrill are less evident, and eventually may disappear, owing to the feebleness of the left auricle and right ventricle. There are then left a short, sharp first sound at the impulse, but with no murmur, and a very faint or even absent second sound. With failure of compensation, the accentuation of the pulmonary second sound disappears also.

**Course of the Illness.**—The duration of this stage of ruptured compensation varies greatly, sometimes all the unfavorable symptoms appear step by step, or, on the other hand, only a few develop, and then treatment arrests the progress, and compensation is once more established. With children as with adults, these improvements may only be illusory, and in such cases, after a short stationary period, the downhill course recommences in spite of every remedy.

**Diagnosis in Rheumatic Heart Disease.**—The diagnosis of the various forms of rheumatic heart disease rests upon an accurate study of the physical signs and symptoms which have been described and it only remains here to write a few words upon the general diagnosis of the condition.

This is based upon a study of rheumatic fever. There can be no doubt that rheumatic fever is the most common cause of heart disease in early life, and when confronted with a case, the history of which affords no guidance, but the nature of which appears in no way unusual, it is the safest hypothesis to look upon the condition as due to rheumatism. This cause, however, should not be assumed without careful investigation. Inquiries should be made into the family history, and into the occurrence of repeated tonsillitis, growing pains, and erythematous and subcutaneous nodules must be sought for. Chorea is usually rheumatic in origin, and even if there is no history of rheumatism it may, nevertheless, be the first cardinal evidence of the disease. Clinical experience has clearly shown that a child who has suffered from chorea, with dilatation of the heart, may a year later come under treatment with an endocarditis or pericarditis which is certainly rheumatic.

I am also of opinion that many of the mysterious cases of mitral stenosis which have no history of rheumatism are rheumatic in nature.

Malignant endocarditis is always a difficult problem. There is now a good deal of evidence to show that rheumatic fever is, in many cases, a factor, but it is uncertain whether the majority of those in which there is a previous history of rheumatism are due to rheumatic infection alone, or to this coupled with some secondary infection.

Thus the general diagnosis of rheumatic heart disease is based more upon a study of rheumatic fever than upon the actual condition of the heart itself.

**Prognosis in Rheumatic Heart Disease.**—There are, unfortunately, some great difficulties in making a prognosis in rheumatic heart disease. It

is not a question of calculating the extent of mechanical defect, but a complex problem in which the liability to repeated attacks of rheumatism is most important. We have not yet sufficient knowledge of the laws that govern a rheumatic infection; nevertheless, some facts are known which may be useful in assisting one to give a prognosis. The double inheritance usually implies a liability in the child to severe rheumatic heart disease. The younger the child, the worse the outlook; the poorer the circumstances, the less the hope of avoiding the predisposing causes. These, then, must be taken into account.

In considering *acute* rheumatic heart disease, carditis is the most dangerous to life, mitral incompetence the least, with the exception of the early dilatation, from which there may be complete recovery.

The insidious cases of persistent rheumatic fever, in which many of the rheumatic lesions make their appearance one after another, are most grave, and as Dr. Cheadle has emphasized, when, in such cases, nodules appear, the outlook is very gloomy.

It is a common experience in England to see these cases drift slowly down hill. They may rally for some months, for a year perhaps, or even longer, but even when at their best there is a history of frequent pains in the muscles and limbs, which tells the tale of a persistent enemy.

Acute pericarditis is rarely fatal when it is a first attack, and acute simple endocarditis never; when, however, the heart is already damaged, pericarditis is a very serious matter indeed. The details of the prognosis in such cases are dealt with under the section on Acute Rheumatic Heart Disease (p. 695).

The prognosis in *chronic heart disease* is surrounded with pitfalls. There is no doubt that slight regurgitant mitral lesions are often completely compensated, and leave the heart almost as sound as before, and even slight aortic lesions may disappear.

Mitral stenosis is more grave, because it is exceedingly difficult to say that the lesion is really arrested, and not slowly progressive. If there is no reason for believing that it is advancing, and the absence of symptoms shows that it is well compensated, a useful life far beyond childhood is to be expected. Yet there are dangers in young adult life, especially connected with childbirth, which cannot be overlooked. Mitral stenosis is clearly incurable.

In estimating the amount of damage that has resulted from a valvular lesion, the symptoms must be first taken into account, and then the extent of the lesion be gauged as accurately as possible by the amount of hypertrophy and dilatation of the heart, and by the character of the sounds of the heart. It must not be forgotten that a loud murmur does not, by any means, imply a severe lesion, but that its duration, and the extent to which it encroaches upon the cardiac sounds are of more importance. Combined aortic and mitral lesions are of bad prognosis.

It will be remembered that adhesions of the pericardium are not easy lesions to diagnose; and so it will not be hastily concluded that

because there has been an attack of pericarditis, such a complication has necessarily supervened, or that, in the event even of there being pericardial adhesion, that the condition is necessarily a very grave one. When, in addition to the pericarditis, there has been pleuropericarditis and mediastinitis with adhesion, the prognosis is very serious, for the heart of the growing child is hampered at every beat.

Each failure in compensation leaves the heart at a lower level of efficiency.

Lastly, the physique of the child influences prognosis. The fragile, fair-haired children, with small limbs and frame, are bad subjects and they need very gentle treatment; strong drugs upset them, and heroic measures alarm them. It is my firm belief that the prognosis in such cases is more grave if the rheumatic heart disease is treated as an enemy to be overcome by fierce blows, and the natural processes of recovery placed in the background.

#### UNUSUAL TYPES OF RHEUMATIC HEART DISEASE.

**Malignant Endocarditis.**—Malignant endocarditis is one of the most important forms of endocarditis. The condition variously termed malignant, ulcerative, or septic endocarditis is rare in childhood, but becomes more common in early adult life. Many infections may cause this endocarditis, and sometimes more than one micro-organism has been isolated from the damaged valve. The usually accepted view of the condition when occurring in rheumatism is that it is the result of a secondary infection of the damaged valves. In 1902 Dr. Paine and I showed that the rheumatic infection might, without any added infection, produce malignant endocarditis in man and animals, and for this reason it is considered here among the unusual forms of rheumatic infection. The relation of rheumatism to malignant endocarditis is, according to this view, quite a consistent and rational one and can be stated thus: All micro-organisms which attack the cardiac valves may produce this type of endocarditis, and among the most important of these is the micro-organism of rheumatism.

**Pathology.**—The essential feature is the presence of the micro-organisms in great numbers in the vegetations on the valves. These vegetations are often large and extend from the valve or valves on to the surfaces of the auricles or ventricles, on to the chordæ tendineæ, or on to the commencement of the aorta. In the most rapidly fatal cases the vegetations are small, yet enormous numbers of the infective agent are found in them (Fig. 154).

When the relations of these vegetations to the current of the blood stream is recognized, it is at once apparent that the infection must be carried all over the body, sometimes in the form of detached fragments of the infected valve, at other times as the micro-organisms themselves which fringe the border. Whatever the cause of the malignant endocarditis may be, it is apparent that the systemic infection must be very



severe, and that the material particles detached by the blood stream will give rise to those important secondary lesions termed infarcts.

In rheumatic cases the mitral valve is most usually affected, though the aortic and tricuspid are sometimes damaged.

The figure (154) shows a good example of the change produced in the valves in man. As a rule, the valve that is attacked has been injured by previous endocarditis; but the interpretation sometimes given, that the micro-organisms prefer a damaged valve, appears to me less likely than that the resistance of the patient has been lowered by previous infections. Then the vital tissues of the valve are less able to cope with the fresh attack, or it may be they harbor the bacteria in a resting stage.

It is essentially a local process—therein the valve is the breeding focus of the disease—and if this could be cut out one feels the illness might be arrested. In childhood we know that the tendency of severe infections is to generalize, not to localize, and in this fact probably lies in part the explanation of the comparative rarity of malignant endocarditis in the young.

In a series of 15 cases, 8 were certainly rheumatic children; 2 were associated with empyema; 1 with pyemia from suppurative arthritis; 1 with tuberculosis of the lungs; 3 were of doubtful origin.

The morbid anatomy of the 8 rheumatic cases was of the same type. The chief features were: more or less damage of the heart by previous rheumatism and the presence of vegetations of considerable size upon the mitral or aortic valves, and endocardium of the left auricle. Infarctions were found in the kidneys, spleen, brain, and lungs, but never any abscesses. The spleen was sometimes much enlarged without the presence of any visible infarction.

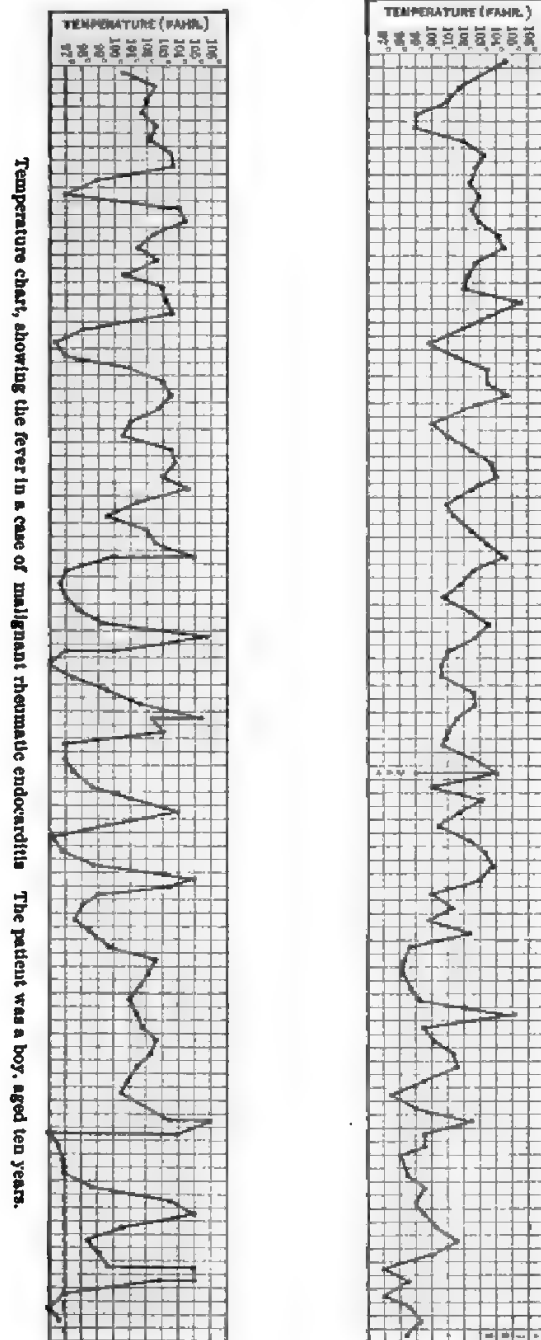
There may be nephritis, the condition resembling that of large white kidney. In 3 cases cerebral hemorrhage occurred from infection of the cerebral vessels, and in 1 an enormous aneurysm of the right common femoral artery developed.

**Symptomatology. Clinical Course.**—The course of the illness is usually prolonged. The onset is sometimes gradual, when shortness of breath, precordial pain, and anemia are often the first warnings, or the illness may follow upon an attack of rheumatism with vague symptoms against which the patient has struggled. With an acute onset there may be shivering and vomiting. The temperature is irregular; sometimes within twenty-four hours the range is considerable (*vide* chart, Fig. 155) in other cases there is a constant degree of pyrexia; finally, in still others, fever occurs at irregular intervals. The pulse is rapid and the heart is excited, and there is often a loud systolic bruit to be heard over the whole precordium. Progressive anemia is generally a striking feature, as are also sudden attacks of pain associated with the formation of infarcts in the different organs. Step by step the child loses ground, and toward the close of life is delirious at nights. The wasting is pronounced, while diarrhea still further drains the patient's strength.

Purpura and increasing breathlessness from the anemia and gradu-



FIG. 155



Temperature chart, showing the fever in a case of malignant rheumatic endocarditis. The patient was a boy, aged ten years.

cardiac failure are warnings of the end, which may come suddenly from cerebral hemorrhage or syncope, or may be gradual from exhaustion of the child's strength.

**Diagnosis.**—In the early stages this is difficult, and the more so because there are transitional forms; these are cases which for a time show malignant symptoms, but eventually quiet down and then the diagnosis is usually thought to have been mistaken.

Sometimes there is doubt at first whether the illness is not typhoid fever, tuberculosis, pyemia, or malaria, but in most cases in childhood the problem is this: Are we dealing with a severe case of rheumatic carditis, are we dealing with the malignant form of rheumatic endocarditis, or are we dealing with some other form of malignant endocarditis? The chief diagnostic points of malignant endocarditis are:

1. The insidious mode of onset, with the early signs of severe injury to the heart.
2. Anemia.
3. Irregular fever.
4. A persistently excited action of the heart, with a loud cardiac murmur.
5. Alteration of the character of the murmurs has not appeared to me of real value.
6. Enlargement of the spleen.
7. Evidences of infarction, including purpura.
8. The occurrence of aneurysms or cerebral hemorrhage.
9. Diarrhea.

These are symptoms which point to the malignant type of endocarditis. The diagnosis of the rheumatic origin is confirmed by the isolation of the diplococcus from the blood and the production of experimental rheumatic fever in animals. On the other hand, some cases may remain doubtful to the end, for it is rarely that one can isolate this micro-organism from the blood during life in these cases.

**Prognosis.**—This is extremely grave. There is little doubt that there are cases which show many features of the malignant type and yet eventually recover; still the recovery is but very imperfect. Those cases which are unmistakably malignant always end fatally.

**Treatment.**—Treatment is exceedingly unsatisfactory. I have personally never seen any good result from the use of antistreptococcic or other sera, though there is more hope in this direction than in any other.

Every effort should be made to maintain the strength and alleviate the symptoms. If the time of year and circumstances permit, I prefer to treat these cases in the open air. Lying on a couch and carefully guarded against draughts, they rest quietly in a pure atmosphere and are, as far as possible, cautiously fed up. The principles adopted for tuberculosis are, in a modified way, applicable to these patients and have the same end in view—viz., to increase the resistance of the body in the hope of mastering the infection.

**Acute Rheumatic Myocardial Failure.**—These rare cases need a few lines of description. The effect of rheumatism upon the myocardium has been already dealt with; this, however, is a condition of acute failure which deserves special mention.

<sup>1</sup> Since writing the above various cases have been recorded in which serum treatment has been thought successful. The serum that has been used has generally been polyvalent and no specific antirheumatic serum has been employed.

The first organ attacked may be the heart, but in some cases, at least, arthritis has preceded. The chief symptoms are: A continued moderate anorexia, a rapid pulse (120 to 140), and rapid respiration; pain over the heart, livid pallor, slight edema, restlessness, and vomiting are also usually present; fainting attacks are liable to occur and death may be sudden or gradual, with complete failure of the pulse at the wrist.

Such cases will remind the reader of the acute cardiac failure in diphtheria (*q. v.*).

**Diagnosis.**—Diagnosis rests on the occurrence of rheumatism and the reponderating evidence of myocardial failure. Antemortem thrombosis may complicate the condition.

The *physical signs* are mostly negative.

The impulse is diffuse and the cardiac area enlarged in all directions, but there is no evidence of pericarditis. As a rule there is a soft murmur, systolic in time, audible over the region of the impulse; this is sometimes conducted toward the axilla, sometimes not. The first sound is short and the pulmonary second sound accentuated and reduplicated. Thus the physician discovers dilatation of the heart with grave constitutional symptoms and infers that the condition is one of acute myocardial disease the result of rheumatism.

The course of the illness is variable, for it carries with it the danger of sudden death. Should the illness prove fatal, as it may within a week from the development of the severe symptoms, death may be quite sudden. These very acute cases are generally fatal.

**Treatment.**—I warn against the use of salicylates and should adopt the same methods as are detailed under the treatment of cardiac failure due to diphtheria. (See page 743.)

**Multiple Serositis.**—Another unusual type of rheumatic heart disease is that with which there is associated inflammation of other serous membranes in addition to the pericardial. It is variously named indurative mediastinopericarditis, or, more shortly, multiple serositis. Rheumatism is not the only cause; tuberculosis, scarlet fever, and, possibly, syphilis may also produce the condition.<sup>1</sup>

I have on several occasions made necropsies upon cases of rheumatic fever in children, in which, in addition to acute pleurisy and pericarditis, there has been local peritonitis around the liver and spleen, and once during life have heard in such a case loud peritoneal friction. Three times with Dr. Paine I have demonstrated and isolated the micrococcus from the peritoneal exudation, and once produced, by intravenous inoculation of a rabbit, peritonitis. These, it is true, were acute conditions, but they clearly have a bearing upon rheumatic multiple serositis and afford additional evidence in favor of its occurrence. When these multiple inflammations, instead of being acute, are slow and smouldering, then the clinical picture of multiple serositis will appear.

<sup>1</sup> A valuable account of the disease has been recently given by Dr. A. O. J. Kelly in the Transactions of the College of Physicians of Philadelphia, 1902.

This case, for which I am indebted to Dr. F. G. Penrose, will give a general idea of the type.

A boy, aged seven and a half years, with a rheumatic mother, got through and chilled. A week afterward he developed rheumatic fever and heart disease. Three months later swelling of the face and abdomen noticed. The boy was well nourished, with a fresh complexion and in distress. The veins in the neck were full. The cardiac area was increased upward, and though there was no bruit, the sounds were muffled, and no impulse could be seen or felt. The air entry into the right lung impaired, and the percussion note dull at the base. The urine natural, there was no edema of the lower extremities, but the abdomen was tense with fluid. After tapping, a large, smooth liver was detected. The ascites recurred and he has needed tapping every week up to the time of writing. Although not distressed he is slowly losing ground.

Ascites was clearly the prominent symptom in this case, but, as Dr. Kelly has emphasized, the development of the symptoms differs with the particular serous membrane which is first affected. I think it is reasonable to expect ascites to be the most frequent occurrence, because the pleural and pericardial sacs may be obliterated, but not so the peritoneal, which has to bear the stress of chronic inflammation and cardiac weakness. The course of the illness is prolonged, lasting many months.

**Diagnosis.**—Such a condition is puzzling, for the original illness may be ill-defined and the pericarditis perhaps overlooked. Cirrhosis of the liver and tuberculous peritonitis are easily confused. The following points are of assistance, viz., the evidences of an adherent pericardium, the presence of pleuritis or an adherent pleura, the occurrence of frequent attacks of pain in association with perihepatitis, and the discovery of a large, smooth liver. Some further allusion is made to the subject under Tuberculosis of the Heart.

**Prognosis.**—This is very serious, for several vital organs are attacked. The adherent pericardium cripples the heart, the adherent pleura encases the lung, and the ascites impairs the functions of all the abdominal viscera. Lastly, the repeated paracentesis undermines the patient's strength by drawing away gallons of albuminous fluid. It is that the course is a downhill one.

**Treatment.**—Treatment is palliative; paracentesis being necessary when there is much discomfort, or when the fluid in the abdomen embarrasses the action of the heart or lungs.

#### SOME COMPLICATIONS OF RHEUMATIC HEART DISEASE.

**Infarction.**—Allusion has already been made, under Malignant Rheumatic Endocarditis, to the occurrence of infarctions. These may be met with in cases which are looked upon as examples of simple rheumatic endocarditis, in association with antemortem thrombosis. This thrombosis, as experiment has shown, may occur from myocar-



weakness without any visible endocarditis. Infarctions may also occur without either evident antemortem thrombosis or endocarditis. Infarcts, then, may be caused by bacterial emboli and by detached fragments of vegetations, by blood clots formed in the ventricles or auricles, and, lastly, they may be encouraged by the increased tendency of the blood to form clots, as a result of the rheumatic processes.

No detailed description of the morbid changes will be given, but it is of cardinal importance to recognize that some infarcts contain numerous bacteria, while others contain few or none at all.

They are classified, according to their colors, into the hemorrhagic or red, and the white. In rheumatism they may soften, but do not suppurate, and, as a rule, the necrotic areas in the tissues heal by a process of scar formation. They are met with most frequently in the kidneys, spleen, lungs, and brain, in rare instances in the liver, occasionally in the arteries, and I have once seen an infarct in the pancreas.

**Symptomatology.**—Infarcts may occur in the kidneys and produce little disturbance; nevertheless it will be useful to give some indications—somewhat arbitrary ones, perhaps—which will assist the diagnosis of infarction:

1. *Renal Infarction.*—(a) Sudden pain in the loins, and pyrexia. (b) Sudden hematuria and albuminuria, and the passing of blood and epithelial casts in the urine. (c) Tenderness over the kidney.

2. *Splenic Infarction.*—(a) Sudden pain in the left side, and pyrexia. (b) Splenic enlargement. (c) Splenic tenderness. (d) Occasionally, a friction rub over the splenic area.

3. *Pulmonary Infarction.*—(a) Sudden pain in the chest, and pyrexia. (b) Cough, dyspnea, and hemoptysis. (c) The signs of an area of solid lung in the position of the infarct and sometimes pleural friction over that same area.

4. *Cerebral Infarction.*—(a) A sudden "stroke," with or without complete loss of consciousness, and pyrexia. (b) Paralysis, usually of the hemiplegic type, with or without aphasia.

5. *Infarction in the Mesenteric Vessels.*—(a) Sudden abdominal pain, with more or less collapse. (b) Melena. (c) Meteorism, and sometimes peritonitis.

The occurrence of infarction must always raise the suspicion of malignant endocarditis, and then the prognosis is very gloomy. Cerebral infarctions are the most dangerous; when terminal arteries are obstructed softening of the brain in the damaged area will follow, but when this is not the case there may be good recovery. Infarcts in the lungs, kidneys, and spleen may heal, but scarring and contraction of the damaged areas must follow.

**Treatment.** The great indication for treatment is the pain. This is eased by the use of some preparation of opium and external applications.

**Thrombosis of Veins.**—This is a rare complication, although Schmidt, Letulle, Gatay, and other French writers have devoted considerable attention to the subject.

One of these cases, a girl aged nine years, is briefly quoted below:

The girl was admitted to St. Mary's Hospital under Dr. Chandle, February, 1898, with advanced rheumatic heart disease, following an attack of scarlet fever two years previously. She was a child of rheumatic parentage. Aortic and mitral endocarditis, and pericarditis were present, with edema, cyanosis and nephritis. The occurrence of rheumatic nodules stamped the case as one of the most severe type. In March, there was pleurisy upon the right side and after this some slight improvement. In April, the thrombosis commenced; on the 13th the left side of the neck swelled, and on the 16th the right side. The face now became purple, the lips and eyelids swollen, the neck tense and tender. Her temperature was subnormal. On the 17th the right arm began to swell, later the left arm and the upper part of the chest. On the 20th two firm cords were felt on the neck. Death occurred on the 21st.

The necropsy showed antemortem thrombosis in the two innominate, subclavian, internal and external jugulars, and axillary veins and also in the inferior thyroid vein. The superior vena cava was filled by antemortem thrombus in the upper two-thirds of its extent.

Six examples have come under my notice, four of which have occurred in children suffering from advanced rheumatic heart disease.

Some writers mention that this form of thrombosis is most common in the lower extremities, but all the cases I have met with have commenced in the large veins at the root of the neck, thence sometimes spreading along the subclavian and axillary vein, at other times spreading upward.

**Diagnosis.**—Diagnosis is based chiefly on the local appearance and spread of the edema, the tenderness along the veins, and pain on movement of the affected part, and the dilatation of the venous tributaries which supply the damaged veins. Fever may accompany the process, and petechie and erythematous patches have been noted.

When the vein is felt as a firm cord, the diagnosis is certain. In difficult cases the swollen face of renal disease and parotitis must be excluded. The prognosis in such cases is grave, for the heart disease is often severe, and there are also the added dangers of a clot being detached from the vein, or of an extension of the process of thrombosis to the right auricle.

The mild cases, and those in which the condition of the heart is not hopeless, may recover completely from the thrombosis.

The correct explanation of the occurrence is doubtful.

It is, in all probability, an active rheumatic process and not merely the result of a failing heart; but whether there is a primary phlebitis and a secondary formation of clot, or whether the thrombosis is the primary change, is not yet clear.

**Treatment.** Treatment is palliative. Pain is relieved by fomentation and by giving opium. The limb is kept at rest, and the movements of the neck controlled, so far as it is possible to do so. When all the acu-

symptoms have passed off, and if the limb still remains edematous, skilled massage is helpful.

**Edema of the Face.**—One peculiarity of the heart disease of childhood is the comparative frequency with which slight edema of the face occurs. An appearance simulating that seen in renal disease is the result, but without any of the changes in the urine usually found in that disease. Thrombosis of the internal jugulars, as already described, is one cause of the edema, though a very rare one; another is indurative mediastinopericarditis, and a third, I suspect, is some slight renal damage.

**Pulmonary Complications of Heart Disease.**—These complications are important because of their frequency and their detrimental influence upon the course of the disease.

**Pleuropericarditis and Pleurisy.**—These are usually true rheumatic manifestations. Pleuropericarditis is detected by a peculiar physical sign, the pleuropericardial friction rub. This sign is heard over those regions of the front of the chest which correspond to the positions where the lungs overlap the heart. The peculiarity of the sign is its double rhythm; not only is it synchronous with the respiratory movements, but it is modified also by the cardiac movements. The respiratory rhythm is the more superficial and the more striking, so that the cardiac rhythm, which is the fainter, may be overlooked. The sign is an important one, because pleurocardial friction may be present without necessarily inflammation of the apposed pericardial surfaces, and thus, although a cause of pain and distress, it is not in itself a danger to life.

Rheumatic pleurisy is a frequent occurrence. The exudation is seldom extensive unless there is in addition tricuspid regurgitation, and then the effusion is in great part a passive one.

It is a cardinal rule of treatment to deal promptly with pleural effusions when they complicate heart disease. Early paracentesis is required. The important indications are: distressing shortness of breath, troublesome ineffectual cough with blood-stained expectoration, and absolute dulness with loss of breath sounds over a considerable area of the lungs at one or both bases. As the heart is already enlarged by disease, it is difficult to estimate the amount that it has been displaced by the effusion.

**Pneumonia.**—Bronchopneumonia is one of the manifestations of rheumatic fever and in the worst cases of rheumatic carditis is a great danger. The temperature is sometimes unusually high (104° F.) for a case of rheumatic fever, and the physical signs are more extensive than the actual lesions in the lung would lead one to suspect. This condition should not be confused with the hypostatic congestion of the lower lobes of the lungs which occurs in severe tricuspid regurgitation. Rheumatic bronchopneumonia may occur early in the illness; the symptoms are acute and the lesion not confined to the bases of the lungs.

**Edema of the Lungs.**—This rare complication, of which I have seen three examples, is most liable to be met with in severe carditis. I am not sure that salicylate of soda is quite free from blame as a cause of its occurrence, but this is only a suspicion. It is a condition comparable to the edema of renal disease and begins, as does that, in the upper



lobes and not at the bases of the lungs. It is not, then, a passive edema due to slow cardiac failure.

In the three cases under my own observation one case recovered and the other two died in twenty-four hours. The indications for treatment are free stimulation of the patient and the interdiction of all depressing remedies.

**Other Complications.** **PURPURA.**—Purpura when it complicates heart disease should always suggest the malignant type of the disease, but it may certainly occur with simple rheumatic carditis. In malignant cases the purpura may occur in all the serous membranes as well as in the skin.

**NEPHRITIS** is also suggestive of malignant rheumatic heart disease, for it is only the graver types of rheumatic fever which so injure the kidneys as to give rise to an acute nephritis. The condition must be distinguished from infarction, which is sudden in onset and accompanied by pain, but not by edema.

**HYPERPYREXIA** and **GANGRENE** of the extremities are both very rare complications.

#### TREATMENT OF RHEUMATIC HEART DISEASE.

The treatment of rheumatic heart disease is not satisfactory, for although much may be done to palliate the condition there are, at present, no means of arresting the scarring consequent upon repair.

**Prophylaxis.**—With the demonstration of the infective origin of rheumatic fever there should be a bright future for prophylaxis. The logical step is to deal with rheumatic fever as with any other great infection and to inquire closely into the laws that govern its occurrence. In time this forward movement must be made, and rheumatic heart disease will become, I have little doubt, less frequent.

There are clear indications to re-examine such problems as the influence of crowded towns, damp houses, malsanitation, and the influence of soil and climate.

So far as the child of rheumatic parentage is concerned, I think it very advisable that a close inquiry should be made into the condition of the place of residence. It should be thoroughly dry, and, if possible, a clay soil is best avoided and a gravel one chosen. A warm, dry, air and equable climate is the best, while bleak winds, sultry heats, and much dust are detrimental. It is not likely that the majority of these children can thus be accommodated, but it is well to bear in mind that cold, damp, and crowded, stuffy rooms are especially to be avoided, for they lend to chills and sore throats. Damp clothes and damp beds hardly need a mention, except to emphasize the fact that errors of this kind in the case of a rheumatic child may prove fatal. In character these children are often unusually bright, emotional, and energetic; they tire their bodies before they tire their minds, and this should be thoroughly recognized by the parents. Discipline, enforced rest after the milder



meal, and early hours, are very valuable to such children, more especially when they are becoming nervous and thin.

The digestion and bowels need careful supervision, for they are often disordered, and then there follow night terrors and insomnia, urticaria, and migrainous headaches. Such ailments should be treated by mild remedies. The old-fashioned rhubarb and soda mixture, preceded perhaps by a small dose of calomel, or a dose of compound rhubarb powder (Gregory's powder), which is best given with a little sal volatile, (ammonium carbonate), and some carbonate of magnesia or citrate of potash for the urticaria.

For deranged digestion with constipation the following is useful:

<b>R</b> —Pulv. rhel. . . . .	0.13 gm.	(2 gr.)
Sodii bicarb. . . . .	0.63 gm.	(5 gr.)
Syr. zingiberis . . . . .	0.65 c.c.	(10 minims.)
Aq. menth. pip. . . . .	q. s. ad 8.00 c.c.	(2 drachms.)—M.

**Sig.**—Three times a day between meals for a child of seven years.

As a tonic, quinine or the alkaline preparation of arsenic may be given, or, better still, a change of air is recommended. Strong doses of iron or large doses of cod-liver oil usually do harm.

The gums and teeth should be looked after and the development of a chronic gingivitis from decayed teeth thus prevented.

As a general tonic, the following is recommended:

<b>R</b> —Liq. arsenicallis . . . . .	0.13 c.c.	(2 minims.)
Tinct. nucis vomice, B. P. . . . .	0.20 c.c.	(3 minims.)
Syr. aurant. cort. . . . .	1.30 c.c.	(20 minims.)
Aq. chloroformal . . . . .	q. s. ad 8.00 c.c.	(2 drachms.)—M.

**Sig.**—Three times a day after meals for a child of seven.

The throat will need especial care, for one certain path of invasion is by way of the tonsils. I teach rheumatic children to learn to gargle early in life, at first with plain water, and later with a gargle of borax, oxymel, and rose-water. It is a great mistake, I believe, to force these children with much study; and public schools of American cities and the board schools of England with their medals and enforced attendances are responsible for a good deal of chorea and heart disease. The resistance of the child is lowered, and then follows a sore throat, with rheumatic heart disease.

The diet should be plain and varied, and there is no objection to the giving of butchers' meat in a limited amount, say once a day.

Warm clothing is very necessary; woollen undergarments for winter and the best quality of interwoven wool and silk for summer; good boots and warm socks and stockings must be insisted upon. There is always this hope to stimulate us, that if we can tide the rheumatic child over his youth, he will become later in life less susceptible to cardiac rheumatism.

**Acute and Subacute Carditis. Medicinal Treatment.**—The most successful method of treatment of the acute phases of rheumatic carditis is a debated question, and at the present time the use of salicylates, in some form or another, has taken such a hold on the medical profession that it will be advisable to comment upon it before turning to the general measures.

Allowing that salicylate of soda is an antidote, can it be safely given in large doses? These are necessary, for even the advocates of a specific action admit that large doses are needful to control cardiac rheumatism.

My own answer is in the negative. I do not think it is a direct antidote for cardiac rheumatism or that it can be given in large doses to children without considerable risk. I cannot accept the statement, and it seems to me only a statement that salicylate compounds are a direct antidote to rheumatism. It is very doubtful that a disease such as rheumatic fever forms only one poison; indeed, such evidence as there is points to it forming many; nor do I think that in the tissues the salicylate compounds have much antibacterial effect, for active rheumatic lesions develop, even when large doses are given.

In rheumatic heart disease there is no doubt that the articular pains are greatly relieved and the temperature lowered by this treatment, but the articular pains—important though they are—are only an incident, and the fever is very rarely a real danger. On the other hand, small doses do not seem to do harm, and certainly relieve the articular pains.

The risks are an idiosyncrasy which may show itself after a very small dose of the drug, in severe vomiting, great depression, and general illness. As I pointed out in the article on rheumatic fever in the *Encyclopedia Medica*, 1901, it may also cause a curious condition of dyspnea resembling that seen in diabetic coma. It is also a cardiac depressant, and, as it produces polyuria, is possibly an irritant to the kidneys.

It does not seem to me that the cases treated by large doses of salicylate of soda do better than others, but rather do worse, for apart from the effect of the drug are apt to arise and are added to the natural anxieties of the disease; nor are cases so treated protected from relapses.

At the present time there appears to me no good reason for adopting more than mild and palliative measures in acute rheumatic heart disease. Possibly in the future it may be necessary to withdraw this statement, and I would do so willingly now if I could see any decisive evidence in favor of a specific treatment.

**The Palliative Management.**—With this method the physician confesses that there is no medicine with a directly curative action, but, keeping before him the great natural resistance there is to the rheumatic infection and the danger of interfering with such by powerful and possibly useless or even harmful remedies, he endeavors to aid the natural resistance in every possible way.

A good example to take of acute heart disease is acute pericarditis with mitral endocarditis.

*Rest* is imperative, and the child should be kept lying down, unless there is difficulty in breathing when in that position. There is not the same need for the wrapping in blankets as in adults, for these children rarely sweat at all freely. The food should be liquid and consist chiefly of milk diluted with water or barley-water. Beef-teen and chicken-broth may be given, and if the appetite is good and the temperature but little raised, a more liberal diet allowed, such as an egg or a little fish.

made bread and milk, jellies, and the like. Severe cases need very careful feeding with peptonized milk or thin gruels every two hours in the day and every three hours at night, but less severe cases may, I think, be fed more liberally with advantage.

*Stimulants* are valuable when there are pallor and restlessness, and when the pulse is flagging and rapid, and the desire for food failing. When patients have been allowed to walk about before being seen by a physician they are often found quite exhausted. Rest and some brandy will then work wonders. A mild case of rheumatic heart disease does not need stimulants; these should be given in bad cases only, and for a definite purpose. For a child of seven 15 to 23 c.c. ( $5\frac{1}{2}$  to  $5\frac{3}{4}$ ) of brandy will be usually sufficient in the twenty-four hours. Much is written of the detrimental effect of alcohol upon the cardiac muscle, but, used for a short time of need, it seems impossible that it can do any harm, and that it aids sleep and digestion in these cases is, I believe, undoubted. Hyperpyrexia is nearly unknown at this early age.

Arthritis is one of the most common of the definite symptoms. Besides bandaging the joints lightly, salicylates of soda in doses of 0.32–0.65 gm. (5 to 10 gr.), in water flavored with orange, every three hours is effective in relieving the pains.

Salicin or aspirin may be used in similar doses for weakly children, but I have found no great advantage in aspirin, which is best administered

**R.S.**

R—Aspirin . . . . .	0.3 gm.	(5 grains.)
Pulv. tragacanth. comp. . . . .	q. s.	
Aq. chloroformi . . . . .	q. s. ad	8.0 c.c. (2 drachms)—M.

Sig.—For a child of seven years 2 teaspoonfuls (8 c.c.) every four hours.

Nothing seems to ease the pain of pericarditis more effectually than an ice-bag, and the steady advocacy of this by Dr. D. B. Lees has done good service. It serves the additional purpose of keeping the child quiet and it is usually well borne, though I do not advise it when the type of the illness is asthenic, and the temperature normal or sub-normal. The physicians should give strict injunctions as to its use, and it must be removed if the temperature falls rapidly and there is any sign of collapse. The assistance of trained nurses in a case of severe pericarditis is extremely useful. If the chest is tender, the bag must be suspended, and it can be well fixed by passing the neck of the ice-bag through a hole in the flannel under the vest; it must not leak, and the ice should be carefully pounded. Its constant application is the most satisfactory, and for the first twelve hours the temperature should be taken every two hours, but after this at longer intervals. That it has any curative action I am doubtful; nevertheless the pain is relieved and the heart quieted.

Hot-water bottles should be placed near the lower extremities while the ice is in use.

For acute pericarditis I do not advise bleeding, except in rare cases where there is some chronic valvular lesion which has embarrassed the right heart and threatens its arrest from overdilatation. This indication will be considered later.



Leeches to the precordium are indicated when dyspnea is urgent, and four are usually sufficient. Blisters hurt children, and, besides, I do not think they do any real good in acute pericarditis.

The bowels should be opened at first with a small dose of calomel, 1 to 2 gr. (0.065 to 0.13 gm.), to be followed by a morning dose of sulphate of magnesia and sulphate of soda. During the illness strong purging is harmful, cascara or liquorice powder usually sufficing.

For restlessness and insomnia opium is invaluable, and nepochthe combined with some potassium bromide may gain a night's rest for the patient, which is of the utmost value.

<b>R</b> Nepochthe . . . . .	4 minims	(0.15 c.c.)
Potas. bromid. . . . .	5 grains	(0.3 gm.)
Glycerin . . . . .	20 minims	(1.5 c.c.)
Aq. chloroformi . . . . .	q. s. ad	2 drachms (8.0 c.c.) - M

Sig. - Two teaspoonfuls (8 c.c.) at night, to be repeated if necessary, for a child of seven years.

Another useful drug is chloralamide, 10 gr. (0.65 gm.) of the powder dissolved in two teaspoonfuls of brandy and diluted to suit the taste with water.

If there is reason to believe that a considerable pericardial effusion is present, digitalis is not safe, but if there is dilatation and the action of the heart is rapid and excited it is indicated in small doses given both day and night. Neither this drug nor strychnine should be left off suddenly if it can possibly be avoided, for the heart feels the sudden loss, and collapse may follow.

<b>R</b> Tinct. digitalis, B. P. . . . .	5 minims	(0.3 c.c.)
Glycerin . . . . .	15 minims	(1.0 c.c.)
Infus. auranti . . . . .	q. s. ad	2 drachms (8.0 c.c.) - M

Sig. - Two teaspoonfuls (8 c.c.) every four hours for a child of seven years.

Strychnine is much used, but I would repeat the valuable warning given by Dr. Cheadle against its use, either when the heart is excited or at a too early date in the illness. Overstimulation of an excited heart and premature stimulation of a diseased one are serious errors of treatment.

<b>R</b> Liq. strychnine B. P. . . . .	2 minims	(0.12 c.c.)
Sp. chloroform . . . . .	2 minims	(0.12 c.c.)
Infus. auranti . . . . .	q. s. ad	2 drachms (8.0 c.c.) - M

Sig. - Two teaspoonfuls (8 c.c.) every six hours for a child of seven years.

The drug is, I think, best given at first by the mouth and later hypodermically, every six hours.

In sudden collapse from acute heart failure the hypodermic method is most valuable, and at such a crisis a mixture of ammonia and ethyl is useful as a powerful stimulant, and can be given every two hours for three or four doses.

Dilatation of the stomach and vomiting may add to the difficulty of treatment; if they occur it is very necessary to decide whether it is the method of feeding, the medicine, or the cardiac failure that is most to blame. In any case prompt treatment is needed. Milk should be peptonized, or less should be given; concentrated meat essence



in teaspoonful doses will sometimes arrest vomiting if all other food is stopped for twelve hours. In the worst cases nutrient enemata must be relied upon.

Bismuth is indicated when there is irritability of the gastric mucous membrane from enfeebled circulation; it is best given as the subcarbonate and in large doses, bismuthi subcarbonatis, 0.65 to 1.0 gm. (10 to 15 gr.), combined, if necessary, with nepoche. Salicylate of soda and digitalis may both cause vomiting, but the former very rarely does so in the small doses advocated for the arthritis. If the digitalis causes vomiting, it is well to leave it off and to substitute caffeine.

R Caffein.	0.2 gm.	(3 grains.)
Spirit. camphoræ	0.3 c.c.	(5 minims.)
Mucilaginis acacis	1.3 c.c.	(20 minims.)
Aquæ chloroformi	q. s. ad	8.0 c.c. (2 drachms.)—M.

Sig.—Two teaspoonfuls (8 c.c.) every six hours for a child of seven years.

Pulmonary complications must be treated upon the lines laid down in the article dealing with Respiratory Diseases (*q. v.*). It is important, however, to bear in mind that pleural effusions must be tapped early in all cases of heart disease.

Many a case of pericarditis runs its whole course without any indication for very special measures; in such cases quinine given in tonic doses is a useful routine prescription.

Sometimes it seems to me considerable harm is done to a child by continually worrying it with medicine, food, temperature taking, milants, and what not; the child never has a quiet moment, for if there is nothing else to be done his pillow is shaken or the quilt put straight. Rather than this, I would prefer to give no medicines at all, but to trust that under the influence of regular feeding, a comfortable and warm bed and peaceful moments, his leukocytes will quietly destroy the micro-organisms.

The convalescence after carditis is prolonged, so that the key-note in the management of the patient should be caution. There are no hard and fast rules to be followed, but a continued normal temperature, the absence of rheumatic symptoms, the improvement in the pulse, and the diminution in the size of the heart are important guides. The cardiac tonics should be gradually withdrawn, and be replaced by quinine or salicylate of quinine, or a little arsenic in alkaline solution given as general tonics, and the following are suggested:

R—Rheumatin	0.2 gm.	(3 grains.)
Pulv. tragacanth. comp.	q. s.	
Aq. chloroformi	q. s. ad	8.0 c.c. (2 drachms.)—M.

Sig.—Two teaspoonfuls (8 c.c.) three times a day after meals for a child of seven.

R—Ferri et ammonii citrat	0.3 gm.	(5 grains.)
Liq. arsenicali	0.2 c.c.	(3 minims.)
Glycerini	1.3 c.c.	(20 minims.)
Aq. chlorof.	q. s. ad	8.0 c.c. (2 drachms.)—M.

Sig.—Two teaspoonfuls (8 c.c.) three times a day after meals for a child of seven.

Some care is required in prescribing iron for the anemia, because the digestion is easily disturbed; the alkaline preparations, however, can be given with success.

Sitting up, getting on to a couch, putting the feet down—in a word, each forward step will be gauged. Above all let me utter a warning against the sudden dismissal of a rheumatic child from a hospital to return to a poor home, for the purpose of getting an empty cot.

The use of passive movements for bridging over the wide gap between complete rest and voluntary movement is of great practical value.

For the well-to-do, it is advisable, as soon as the journey can be undertaken, to remove the child to a warm, sunny, dry climate, preferably inland, where he can lie flat on a couch out-of-doors for hours, or be wheeled about in the fresh air. Drugs, such as arsenic and iron, can then be given up. There seem to me to be the same indications for the liberal feeding of the convalescent in this disease as there are for the liberal feeding of the convalescent from tuberculosis.

It is wonderful what a stimulus a change of scene and air may be to the invalid whom an overcautious treatment has kept stagnant in one room. True enough of the adult, it is doubly true of the child provided, always, that it is not agitated by many visitors and exciting books, or, as is so often the case with rheumatic children, by high nervous parents.

When walking can be undertaken, the same cautious forward policy should be pursued. At first the child should have steady exercise on the level, then later up gentle inclines, the ordered passive movements now giving place to ordered voluntary movements; in this way the cardiac muscle is strengthened. The care and time over such details are well spent. Some philanthropist should found homes for the children of the poor suffering from rheumatic affections of the heart, where during convalescence, treatment on these lines could be carried out.

**The Salicylate Treatment of Acute Rheumatic Heart Disease.**—This method has been recently detailed by Dr. D. B. Lees, of London. It is in principle the antithesis of the palliative method, and claims to be specific.

Dr. Lees points out that, occasionally, ill effects may arise from the use of the drug, but believes these to be rare. The occurrence of air hunger, he thinks, may be explained by the action of excess of the drug upon the respiratory centre, and this he counteracts by the combination of double the dose of bicarbonate of sodium with the salicylate of sodium. The depressant action of the drug is, in his opinion, greatly exaggerated. Should there be intolerance, after suspension of the drug for a few hours, it should be recommenced in small doses and then progressively increased.

For a child from six to ten years of age 0.65 gm. (10 gr.) of salicylate of soda and 1.3 gm. (20 gr.) of bicarbonate of sodium are given every two hours during the day and every four hours during the night; after a day or two these doses may be increased to 1 gm. (15 gr.) and 2 gm. (30 gr.), respectively, and later to 1.3 gm. (20 gr.) and 2.65 gm. (40 gr.).

The treatment should be persisted in through the attack, and only stopped when all the active symptoms have abated, and then gradually relinquished.

In addition to this, for reducing the cardiac inflammation, an ice-bag is applied to the precordium, and if on careful percussion of the deep cardiac dullness the right auricle is found distended, leeches are applied below the right nipple, preferably before the ice-bag is used.

Dr. Lees maintains (*Harveian Lectures*, 1903) that treatment of this kind "greatly diminishes the tendency to rheumatic relapse, checks the inflammation, increases the vigor of the muscular fibre, and diminishes the dilatation, thus enormously assisting the forces that make for repair."

**Treatment of Chronic Heart Disease.**—When the lesion is compensated, the treatment resolves itself into a discrete study of the general health and careful superintendence of the active pursuits. When compensation is failing, rest in bed is the first indication, and that alone may be sufficient without any further treatment. The failure, however, may be acute, and in mitral disease the right side of the heart, hampered by overdistention, may threaten to fail entirely. Again, when there is well-marked tricuspid regurgitation, the functions of all the organs, and especially the abdominal, are interfered with by the congestion of the venous system. These, then, are both of them important indications for a treatment more drastic than that of rest.

When the pulse at the wrist is small, the child blue and dyspneic, the cardiac enlargement to the right of the sternum much increased, and the epigastric pulsation forcible, it is necessary to abstract blood in one way or another. The more usual method is to place four to six leeches over the tender liver, and afterward to let the leech-bites bleed or not, as may be thought fit. Sometimes, even in children, the urgency is extreme and then the median basilic or external jugular vein should be opened with a lancet. It is a cardinal rule that children bear loss of blood badly, but, in such crises, they bear overdistention of the right side of the heart still worse. The withdrawal of four to six ounces will suffice. When the blood has been abstracted, the relief obtained is very striking; but no time should be lost in rousing the heart to more vigorous action. Strychnine should be administered hypodermically or by the mouth, and when the mitral regurgitant lesion is the chief lesion, digitalis, also, should be given every three hours until the heart has rallied. Stimulants are needed, and the extremities must be kept warm. When the heart has recovered it is not advisable to keep up this strong stimulation, but by degrees to lessen it to a gentle tonic action.

The general congestion of the viscera in the less severe cases depends primarily upon the heart-failure, and when this condition improves the congestion lessens. It is, however, a difficulty and danger in itself, and as such needs treatment. The hepatic functions are sluggish; the stomach is dilated and its mucous membrane catarrhal; the kidneys excrete with difficulty a scanty and sometimes albuminous urine; further, the lungs are congested at their bases. Vomiting, nausea, dyspnea and insomnia greatly add to the distress and militate against recovery. It is well, then, to ease the liver by a dose of calomel followed



by a saline purge, and it is a sound rule in practice to do this before giving a drug such as digitalis, which readily deranges the digestion. Bismuth and soda may be required to soothe the mucous membrane of the stomach; and it may be necessary when there is a tendency to vomit, either to give small quantities of peptonized milk alternately with little meat juice, or even to stop food by the mouth altogether.

Insomnia needs prompt treatment; trional, chloralamide, bromide of sodium or potassium may be sufficient, and if there is no marked cyanosis or renal congestion opium may be used very successfully, either alone or combined with the bromide. In the mean time rest and cardiac tonics should, in a favorable case, be aiding the heart, and in this way the child may be brought to a condition of comparative comfort from one of great distress.

**Treatment of Severe Attacks of Palpitation with Precordial Pain.**—In some cases of mitral stenosis there may be most distressing attacks of pain and palpitation, which are probably due to acute overdistention of the right side of the heart. This condition may be relieved by bleeding, but there is often need in addition for some medicinal treatment to relieve the anguish. Inhalation of nitrite of amyl is sometimes of service, while in other cases a combination of atropine and strychnine injected under the skin or given by the mouth, is the most effective remedy.

R—Liq. strychnine, R. P. . . . .	0.06 c.c.	(1 minim.)
Liq. atropine sulphatis, B. P. . . . .	0.06 c.c.	(1 minim.)
Syrupus aurantii . . . . .	0.60 c.c.	(10 minims.)
Aque chloroformi . . . . .	ad 8.00 c.c.	(2 drachms.)

Sig.—Two teaspoonfuls (8 c.c.) at once.

There is considerable risk in giving morphine in such a condition for there is often a period of shock noticed immediately after a hypodermic injection of that drug; death may occur during this period before the soothing effect of the morphine has time to take effect.

Bromide of potassium is ineffectual. Useful enough for the purpose of soothing the state of nervousness which necessarily follows such an attack, it is too slow in its action, and too feeble in its power to alleviate pain and to cope with the urgency of this symptom.

**Anasarca.**—Although such cases are uncommon, yet, now and again, a child may become very edematous, and this edema will necessitate some treatment. The liquids in the diet should then be diminished as much as is possible, without causing distress. Digitalis is often of much use. The lower extremities can be drained by means of Southey's tubes, introduced with every precaution against septic infection.

When there is ascites, paracentesis should not be delayed if the discomfort from the distention, embarrassment of the action of the heart, difficulty in respiration from upward pressure upon the bases of the lungs, or diminution in the secretion of urine. The fluid should be slowly withdrawn, and the abdominal wall supported by a many-ribbed binder, which is gradually tightened as the fluid is withdrawn.

This general rule can be formulated for the use of digitalis in the



heart disease of childhood: It must be given with great caution, if at all, when there is good reason to believe the cardiac muscle is greatly damaged, or when the muscular contractions are impeded by a large pericardial effusion.

**Serum Treatment.**—The common belief that rheumatic fever is an attenuated pyemia has led to the use of antistreptococcic serum in the treatment of rheumatic heart disease. This method I have tried in carefully chosen cases, but without any success.

An antibacterial serum such as the antistreptococcic is not at present a satisfactory remedy, and I would warn the practitioner against the assumption that it is necessarily innocuous, even if it is not useful. Menzer has recently introduced a special serum, but, not having any experience of its use, I would refer the reader to his original paper, which will be found in the *Zeitschrift für klinische Medizin*, Berlin, 1902.

## CHAPTER XXX.

### HEART DISEASE FROM DIPHTHERIA AND OTHER INFECTIOUS DISEASES OF THE ARTERIES.

#### HEART DISEASE RESULTING FROM DIPHTHERIA.

THE form of heart disease which results from the diphtheritic infection differs remarkably from rheumatic heart disease.

We are dealing in both classes of cases with the poisons of an infective agent; in diphtheria, however, as a rule, the bacilli do not gain a foothold in the cardiac tissues, but, localized to the area of the throat, they produce poisons which circulate in the blood.

There is an atmosphere of tragedy surrounding this condition. For there is brought to our minds the recollection of sudden and unexpected deaths occurring at a time when apparently the acute manifestations of the disease are over. Yet it is my firm belief that the more carefully these cases are observed and treated, the less frequently will these calamities occur, for there is usually some warning that the heart is damaged before the sudden collapse occurs. In becoming acquainted with the experience of others, I have been surprised with the differences that exist in the interpretation of slight disturbances of the heart in diphtheria, but I agree with those who attach great importance to them, however slight and seemingly trivial they may be. Treatment may become overcautious, but it is wise to run no risks in the heart disease of diphtheria.

**Pathology.**—Diphtheria, when it damages the heart, almost invariably does so through its action on the myocardium; it is exceptional to find endocarditis or pericarditis, and should either of these conditions be present it becomes probable that there is a mixed infection.

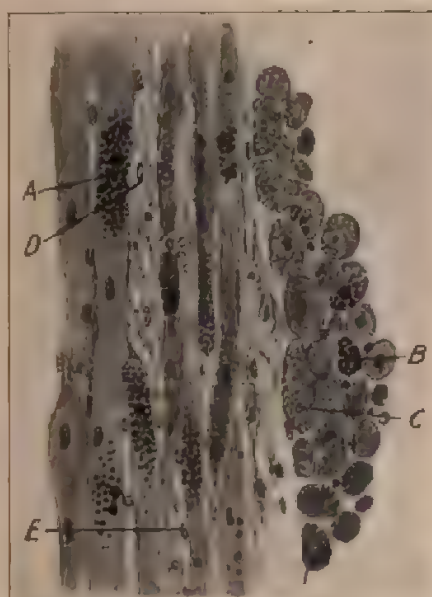
It matters but little from the point of view of practice whether the damage commences in the nerve endings or in the muscle fibres, but it is important to determine whether the poisons attack the vagal centres in the medulla, and thus affect the heart indirectly by lesions at a distance from it. If these centres are rapidly destroyed, then a sudden fatal issue may occur with very slight warning.

In some fatal cases with paralysis of the limbs, changes have been demonstrated in the anterior cornual cells of the spinal cord. Butten is of opinion that the dominant nervous lesion is a parenchymatous degeneration of the myelin sheath of the nerves, while Bolton found degenerative changes in the vagal nucleus in the medulla, in a series of cases reported in the *Edinburgh Medical Journal*, April, 1902.

It also seems very probable that there is direct toxic action upon the cardiac muscle fibres.

In some cases there are very definite changes in the cardiac muscle; changes of such a nature that some considerable period of time must have been occupied in their production. Then again there are many, not fatal, but which resemble these in their clinical features, and so support the view that the cardiac wall is often the seat of the damage. If a microscopic examination is made the muscle is in some cases seen to be profoundly altered. Thus, some fibres are completely destroyed, and in others the nuclei are swollen or shrunken and show hyperchromatosis; the striation of the fibres is lost more or less completely, and their shape is irregular; extensive fatty changes sometimes occur in scattered areas throughout the heart, as shown in the accompanying figure (Fig. 156).

FIG. 156



Myocardial disease in diphtheritic paralysis. A, fatty degeneration of a muscle fibre. B, the same in cross-section. C, degenerating muscle fibre. D, muscle fibre destroyed. E, connective-tissue cell.

The interstitial tissues show little change, though there may be an increase of cellular elements, and minute hemorrhages in the neighborhood of the small bloodvessels.

**Symptomatology.**—I divide these cases into two chief groups, but cannot draw any hard-and-fast line between them.

The first, a small one, contains those dangerous forms of paralysis in which the disease is widespread and implicates, among other structures, the respiratory muscles. In these the condition of the heart is but one element in the danger, and sometimes not the most prominent.

Such cases as these are most serious, and many of them die. It is

for this reason very necessary to study the symptoms that precede the final stages, and the more so because the diphtheria itself may be overlooked. Sometimes for example there is only the history of a sore throat, and then some four weeks afterward the child is noticed to squint, liquids regurgitate, the voice becomes nasal, and there is ataxy. Vomiting may follow, or there may be difficulty in swallowing. A still more alarming symptom is a curious, ineffectual cough, which always means that the diaphragm is weak. Examination shows that the epigastrium does not move forward in inspiration, or that the diaphragm is completely paralyzed and the epigastrium is drawn in with inspiration. The pulse is quickened, of low tension, and perhaps irregular. The cardiac impulse is ill-defined, and there is slight dilatation with, it may be, a systolic apical murmur. Death in such a condition is frequently very rapid, and at the best there must be the greatest anxiety until the paralysis disappears. The causes of death are, usually, engorgement of the lungs from the respiratory paralysis and arrest of the enfeebled heart, or sudden paralysis of the heart itself. But not all die; some recover even when at death's door, and it is the condition of the heart which is of such vital importance that leads me to describe the condition here rather than leave it entirely to the article on Diphtheria.

The *second* group is a larger one and includes those cases in which the cardiac weakness is the prominent symptom, and perhaps the only one. The evidences show themselves usually within the first four weeks and even within the first week after the infection.

If the child is in bed, and that is the rule when the original illness has not been overlooked, the general condition undergoes no striking alteration, though pallor and muscular feebleness are apparent in the more severe cases.

It is the pulse and heart that need careful physical examination.

The pulse is altered; it may be unduly rapid or slow or may be irregular; the tension is low. The changes in the heart are unobtrusive; the impulse is feeble, perhaps irregular; the area, on careful percussion, is slightly increased, especially to the left; the first sound at the apex is short, and there may be a soft systolic murmur, usually heard most clearly within the nipple line; the pulmonary second sound is accentuated, and sometimes a basal systolic murmur is audible. Yet it is very often indeed that no bruit at all is heard. There is no edema, no pain, no startling dyspnea. If the child is running about, fainting attacks occur, and then it is fortunate if paralysis of the larynx supervenes, and prevents—what may be a fatal error—the advice that the child should be sent to the seaside to recover from the debility.

The urine in all cases should be tested for the presence of albumin. When the fatal result is approaching the face is pale, the respiration is sighing, and the extremities are cold, but, as a rule, there is no delirium.

Dr. Villy, in an excellent paper (*Medical Chronicle*, September, 1879), emphasizes the importance of vomiting in these cases. This vomiting is associated with organic changes in the mucous membrane of the stomach, and may precede the cardiac weakness.



The vast majority of cases in this group recover, with careful treatment, but some of them are very slow in so doing, and tachycardia, irregularity, and impairment of the force of the heart sometimes lasts for many months, or even for some years.

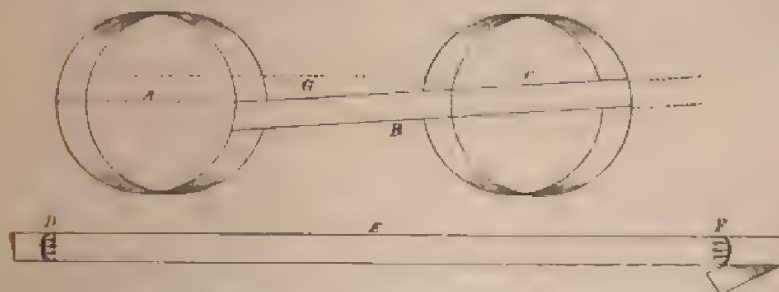
**Diagnosis.**—The diagnosis of heart disease in diphtheria requires care, and the absence of any striking murmurs has led to many mistakes. The danger of an oversight is much increased if the history of diphtheria is not clear.

The method of making outlines, as already recommended (p. 684), is very useful in this condition also, as a check to hasty examination.

Any cause of dilatation of the heart will have to be reckoned with in forming a judgment, but influenza, and some rheumatic cases which damage the myocardium disproportionately, are those which are the most difficult to exclude.

The other evidences of diphtheritic paralysis must be sought for, and cultures taken from the fauces for the discovery of the Klebs-Loeffler bacillus.

FIG. 157



Reins for controlling the movements of a child suffering from diphtheritic heart disease: A, C, arm straps; B, chest strap; D, E, F, strap slipped through A and C, passing beneath the child and buckled to the frame of the bed; G, position of strap, D, E, F.

**Prognosis.**—This is always grave in the severe cases, but in those in which there is only slight dilatation or irregularity it is good. If the heart is damaged in the first ten days the outlook is more serious.

Dangerous symptoms are the occurrence of pharyngeal and respiratory paralyzes, severe vomiting, great pallor, restlessness, and syncopal attacks.

Turning to the heart itself, a rapid, ill-sustained pulse or a very slow pulse is serious, and when the two sounds of the heart are closely approximated this must be looked upon as a sign of the greatest danger.

The extent of dilatation is not a reliable index, for the worst cases may show but little enlargement of the deep cardiac dulness.

**Treatment. General Management.** In the first class of cases treatment is heavily handicapped by the lack of any drug which has a controlling power over the spread of the paralysis, and yet should it spread even a little farther the child must die. The lives that are saved are

triumphs of a skilled management which has kept the strength maintained until the natural recovery commences. Specially trained nurses are invaluable, for they realize the danger of sudden movements, such as the assumption of the sitting or erect positions, and they understand how to feed the child and how to attend to its wants with the least possible disturbance. Useful reins for controlling the child are those shown in Fig. 157.

Solid food can often be given with great benefit in these cases and, if the nurse is skilful, can be persisted with even when the paralysis would seem to counterindicate it. Failing this, nourishing fluids should be given. In some cases these are best thickened with a little arrowroot, in others, even though swallowing is difficult, the liquids are taken better unthickened. Milk, beef-tea, and the yolk of a raw egg may be used, and, if necessary, the milk must be peptonized. Great care should be taken not to hurry the child and thus cause it to choke and aid the supervention of bronchopneumonia by the aspiration of fluid into the air passages.

If vomiting is severe, nutrient peptonized enemata of milk and beef-tea with some brandy or whiskey are needed, but the outlook is very gloomy when these are called upon. Resort may also be had to saline infusions under the skin as follows:

**B**—Normal saline solution.  
The white of one egg.  
(Glucose to make a 5 per cent. solution  
To make 2 ounces (60 c.c.).

Nasal feeding, which in children is usually more successful than the passage of the tube by the mouth, is most valuable if done skilfully, but let me warn against one danger. It may be that the patient has taken but little food by the mouth, for some has been regurgitated and some rejected, and now, when the tube has been passed into the stomach, an effort is made—well-intentioned enough—to cover the deficit by a hastily given, large, and concentrated meal. The result may be most disastrous; the feeble muscular wall of the stomach gives way, the stomach dilates and presses upon the already embarrassed heart, causing sudden death, or, if not that, gravely imperilling the recovery. Stimulants in the form of brandy I give freely, though there is no consensus of opinion upon this point; it should be given to a child of five to ten years as a medicine in a little water, in doses of 8 c.c. (2 dr.) or more every four hours, night and day.

It is a mistake to treat these cases with great energy; they should be examined as little as possible, and their food and medicines arranged so as to prevent continued disturbance. The bowels are better regulated by carefully given enemata than by purgatives. When the diaphragm is paralyzed, it is not advisable to let the child lie persistently on the back, and he should be turned gently on to the side from time to time. Some raise the foot of the bed, thus hoping to aid the drainage of the edematous fluid from the lungs. It is essential to avoid exposure and chilling of the surface, for an added bronchitis will be fatal.

Oxygen is serviceable when the breathing is embarrassed and the color bad, and should then be used continuously.

From such remedies as electricity or leeches to lessen the congestion I have seen no advantage.

**Medicinal Treatment.**—The drug usually relied upon is strychnine. The greatest effect is obtained by hypodermic injection, but it is well to begin first with doses by the mouth. Dr. D. B. Lees strongly advocates its combination with atropine in full doses in such a prescription as the following one:

R—Liq. atrop. sulphatis (B. P.) . . . . .	0.06 c.c.	(1 minim.)
Liq. strychnie sulphat. (B. P.) . . . . .	0.06 c.c.	(1 minim.)
Syrup. aurantii cort. . . . .	0.60 c.c.	(10 minims.)
Aq. chloroform. . . . .	q. s. ad 8.00 c.c.	(2 drachms.)—M.

Sig.—Two drachms (8 c.c.) every four hours.

The atropine is pushed until dryness of the mouth, dilated pupils, and a dry skin are noted.

Improvement shows itself in an evident way by the return of the movement of the diaphragm, increase in the volume and strength of the pulse, decrease in the area of cardiac dullness, and a greater distinctness in the character of the heart sounds.

Unfortunately a relapse may occur, but if not the improvement is slow and steady. No relaxation of precautions must be permitted for a moment until all traces of the respiratory and cardiac paralyses have disappeared, and when the emergency is past the treatment will be such as will be described for the second and less fatal group of cases.

However mild a case of diphtheria may be, it is advisable to keep the child in bed for three weeks after the disappearance of the membrane. Then each step forward is taken cautiously, and a month will have been passed before the heart and pulse are allowed to escape careful observation. It is the last week that needs special watchfulness. The bed-rest, sitting up, lying on the couch, and the first exercise, all these are events in the convalescence which need testing, and irregularity of the heart's action or slight dilatation should delay the advance. The knee-jerks must be tried while the child is in bed, for by their disappearance they sometimes give an early warning of paralysis.

A useful routine prescription is the following mixture of iron and nux vomica, and quinine in small doses is also a valuable tonic:

R—Liq. ferri perchloridi (B. P.) . . . . .	0.3 c.c.	(5 minims.)
Tinct. nucis vomice . . . . .	0.2 c.c.	(3 minims.)
Syrup. aurantii cort. . . . .	1.3 c.c.	(20 minims.)
Aq. chloroformi . . . . .	q. s. ad 8.0 c.c.	(2 drachms.)—M.

Sig.—Two drachms (8 c.c.) three times a day for a child of four years.

R—Quinin. sulphat. . . . .	0.03 gm.	( $\frac{1}{4}$ grain.)
Acid. sulphurici aromat. dil. . . . .	0.13 c.c.	(2 minims.)
Glycerini . . . . .	1.30 c.c.	(20 minims.)
Aq. chloroformi . . . . .	q. s. ad 8.00 c.c.	(2 drachms.)—M.

Sig.—Two teaspoonfuls (8 c.c.) three times a day.

It is not advisable in these cases to use powerful drugs such as digitalis and strophanthus, which alter rhythm of the heart; nor should I advise

the common practice of stimulating the heart with hypodermic injections of strychnine. None of these powerful drugs has a directly curative effect on the cardiac muscle. Strychnine, by getting the full use out of the healthy fibres, is most valuable at a time of urgency, but the tendency is to abuse it.

As a rule the slight disturbances after diphtheria get quite well, and when this is so a change to seaside or country will correct the anemia and general debility. But sometimes the heart does not recover completely for many months. Tachycardia, dilatation, and breathlessness are complained about, and the parents remark upon the great change in the general health of the child since the illness. Time and caution are the two first necessities. The bowels must be regulated and all violent exercises prohibited. Steady exertion, such as a quiet walk every day, will often do good, unless it should be that this causes palpitation and irregularity. Schoolwork, heated rooms, and children's parties should be exchanged for nature's lessons in the country, pure air, and an hour's rest after the midday meal. A good wholesome diet is most important.

So far as I am aware no drug has a curative effect upon these weakened hearts, but general tonics are very serviceable.

#### HEART DISEASE RESULTING FROM SUPPURATIVE INFECTIONS.

**Suppurative Pericarditis. Etiology.**—This is, perhaps, one of the most difficult of the diseases of childhood to diagnose or to treat with success, and though not common can hardly be called rare. There is no doubt that all those microbial infections which produce suppuration can cause suppurative pericarditis, but experience has shown that about 80 per cent. of the total number are associated with pulmonary diseases. This same percentage represents cases of suppurative pericarditis occurring in children under four years of age. With at least 60 per cent. an empyema is associated, while in other cases abscess and gangrene of the lung, mediastinal abscess, suppurating bronchial lymph nodes, tuberculosis, and pneumonia have been recorded. Osteomyelitis and general pyemia resulting from suppurating wounds are the antecedents in a small percentage of the cases.

Measles, influenza, and other infective diseases predispose to suppurative pericarditis, and in addition to respiratory diseases—meningitis and peritonitis have been frequently noted as occurring simultaneously with the pericarditis. The cardiac valves are rarely damaged—a fact strongly in favor of the specific nature of rheumatic fever—and it is quite exceptional to find suppurative pericarditis as the primary lesion.

**Pathology.**—After death the pericarditis may be found either in the earliest stages or well advanced. It may require careful observation to detect a few flakes of exudation, or there may be great distention of the pericardium, with as much as six or seven ounces of liquid pus in the sac. The parietal pericardium is sometimes greatly thickened.

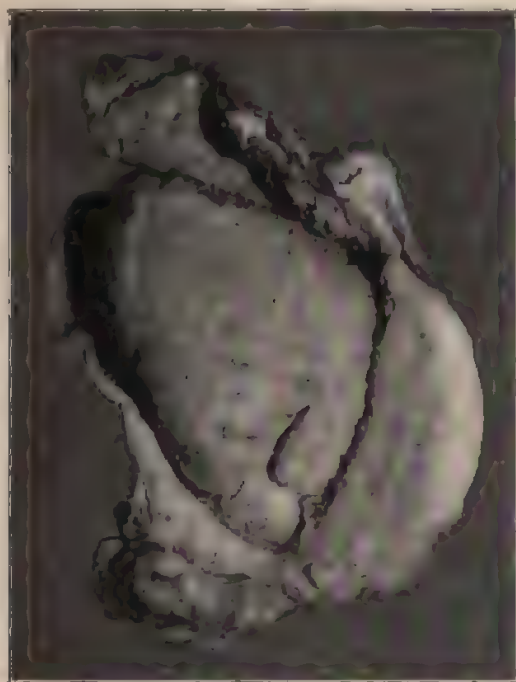


the heart is often not dilated at all, and, except that the muscle substance is somewhat pale, is otherwise normal.

The exudation is sometimes in the form of creamy pus and sometimes there are fibrinous strands, but, as a rule, there is rapid solution of the fibrinous elements and no adhesion (Fig. 158).

In very exceptional cases an abscess in connection with the air passages opens into and infects the pericardium, thus giving rise to pneumopericardium. Since bacteriological investigations have been made, *streptococcus lanceolatus* is the bacterium that has been found

FIG. 158



Suppurative pericarditis. The pericardium is opened; the sac was greatly distended with a purulent fluid.

often in the pus, for it is the micro-organism especially responsible in pulmonary disease. In cases of osteomyelitis the staphylococcus has been isolated, and in some instances the streptococcus species.

In the days when empyemata were not treated surgically, one of the non-terminations was suppurative pericarditis. This points to the possibility of the pericardium being, in some instances, secondary to the pulmonary disease. There are, however, other cases in which it seems clearly that the pericarditis is a part of a general infection of the heart membranes.

**Symptomatology.**—The following cases well exemplify the usual course of the illness and gives a better idea of the general course of the disease than a mere repetition of probable symptoms would do:

CASE I.—J. G., aged nine months, came under observation for cough, wasting, vomiting and diarrhea. The illness had been gradually developing for six weeks. The previous history and family history were unimportant.

There was dulness over the upper lobe of the left lung, also in the upper part of the left axilla and posteriorly down to the level of the inferior angle of the scapula. Over this dull area the breath sounds were diminished and the breathing bronchial. At the right base posteriorly there were numerous râles. The limits of the deep cardiac dulness were: Above, the second rib; to the left, the vertical nipple line; to the right, one-half inch external to the right sternal margin; the cardiac sounds were normal. The temperature was 102° F., pulse 160, respiration 38. The probable diagnosis was tuberculosis. The temperature fell, but still the child lost ground; now two sharp crepitations were detected over the dull area and impairment of resonance over the right lung posteriorly, reaching down to the fifth rib. Death occurred on the tenth day. Necropsy showed empyema between the left lung and pericardium, and also over the upper part of the right lung. There were also bronchopneumonia and suppurative pericarditis with an ounce of pus in the pericardial sac. By bacteriological culture the streptococcus lanceolatus was found.

CASE II.—M. P., aged two years, came under observation for cough, anorexia, and drowsiness. The child was thin, pale and rickety. The prominent symptoms were severe dyspnea and feeble pulse, the rate of which was 168 to the minute. The cardiac dulness was limited: Above, by the second intercostal space; on the left it reached one-half inch external to the vertical nipple line; to the right it reached just below the right margin of the sternum. The heart sounds were feeble. The pulmonary sounds were as follows: There was dulness on percussion and loss of breath at the right base posteriorly, with crepitations at the left apex. The left lung otherwise appeared normal. Exploration of the right pleura discovered pus, which was dealt with in the usual way. There was, however, no improvement. The temperature, which had been irregular, reaching 103° F., continued unaltered, and lividity and syncopal attacks were noted. The child died suddenly nineteen days after admission. Autopsy showed a small empyema in the left pleura and purulent pericarditis.

In neither of these cases was there pericardial friction.

It is apparent that the absence of pericardial friction and the presence of pulmonary disease greatly complicate any description or detection of the pericarditis. When the condition is an incident in a general pyemic infection it may also be overlooked entirely, although, in exceptional cases, loud pericardial friction has established the diagnosis.

**Diagnosis.**—The diagnosis is most difficult because pericardial friction is very rarely detected, and the condition is just sufficiently rare

make the experience of one failure fade from the memory before the next opportunity is presented; nevertheless, there is sometimes such a definite effusion and such evidence of a distended pericardium that the diagnosis is made with confidence, as in this case:

F., aged four and one-half years, was admitted to St. Mary's Hospital, December 22d, suffering from right lobar pneumonia. On admission the impulse of the heart was noted to be below and internal to the left nipple, where it could be both seen and felt. There was persistent dullness at the right base, and on December 31st an empyema was drained. There was no improvement; the child became feeble, cyanotic, and distressed. On January 4th the cardiac area was as follows: Upper limit, second left costal cartilage; left limit, one finger breadth external to the left vertical nipple line; right limit, just internal to the right vertical nipple line. The sounds of the heart were so faint as to be almost inaudible, and over the precordial area of increasing resistance upon percussion. Dr. Pepper opened and drained the pericardium that evening, but the child sank and died twelve days later. This is an exceptional case.

Far more often the attention of the physician is centred on the pulmonary disease, and, to the end, it is supposed that a loculated empyema has been overlooked or that there is a relapsing bronchopneumonia or tuberculosis. Finally, the development of suppurative meningitis may complete the confusion.

This much seems clear, that when in children under four years of age an empyema or bronchopneumonia has been detected, and the course of the disease, in spite of correct treatment, is unsatisfactory, or if pus is liberated from the pleura and the temperature still remains high, and there are wasting and steady loss of strength, then suppurative pericarditis is a probable complication. This is the more likely if the illness has occurred during an outbreak of influenza, or after some infection such as measles. When the possibility of the danger is kept in view, there are other symptoms, more or less equivocal, which may lead to the diagnosis. Great rapidity of the pulse with irregularity, vomiting and livid pallor, panting respiration and orthopnea, with frequent fainting attacks, have all been recorded. When there is progressive muffling of the heart sounds, and, simultaneously, an increase in the area of cardiac dullness, that evidence is exceedingly valuable. Yet it is unfortunate that even this evidence is difficult to obtain; for the pulmonary affection itself, by possibly causing consolidation or collapse, or the formation of fluid in the region of the pericardium, complicates and makes difficult the study of the cardiac dullness. An impairment immediately to the left or right of the sternum, if disproportionate to any impairment elsewhere, is highly suggestive of fluid in the pericardial sac; nor should it be forgotten that the dullness on the left side—due in part to pulmonary collapse—will extend in this condition even up to the left clavicle. Naturally, pericardial friction is always listened for, but is rarely heard.

In a *large pericardial effusion* the pulse is rapid, the wave small, ill-sustained, and sometimes very irregular. There may be some bulging



of the precordial area. The impulse is either absent or an undulating movement may be seen in the third or fourth space on the left side. On palpation, either no cardiac impulse at all is felt or only a distant tap is detected.

The area of deep cardiac dulness is greatly increased and is pear-shaped, with the stalk of the pear in the position of the large blood-vessels.

The change from the absolute dulness over the fluid in the pericardial sac to the pulmonary resonance is abrupt and striking.

The sounds of the heart are distant, or may even be inaudible and give the impression that the heart has been lost.

In young children, collapse of the upper lobe of the left lung may cause the dulness to rise as high as the left clavicle.

**Prognosis.**—The prognosis is certainly very grave. In the cases in which it is part of a general pyemia, death is almost inevitable, and, moreover, until the diagnosis can be more readily made the vast majority of all cases must die. The only hope, at present, lies in surgical intervention, and it occasionally saves the life of the patient.

**Treatment.**—Palliative measures, so far as the saving of life is concerned, are useless, but they promote euthanasia. It is from some active serum that we must look for help, but at present there is no such remedy.

The *drainage* of the pericardium is, therefore, at this time our only resource, and the recent advances in cardiac surgery must encourage us to explore the pericardium with less dread than hitherto.

The surgical measures that can be adopted are two, viz., *paracentesis* of the pericardium and incision and drainage. The first is only of value in those cases in which, owing to the quantity of fluid in the sac there is embarrassment of the heart, yet the condition of the patient is too serious to permit an anesthetic. Then by paracentesis the pressure can be relieved, and an opportunity is given for the circulation to recover. With the improvement in the patient's condition the more radical operation can be undertaken.

In operations upon the pericardium it should be remembered that the level at which the left pleura leaves the middle line, as given by Lauscha—at the fourth costal cartilage—is only correct in a small proportion of cases. As a rule, the left pleura does not leave the middle line until the level of the fifth or sixth costal cartilage is reached.

Paracentesis, as Roberts has pointed out, is most safely performed by the introduction of the needle in the left costoxiphoid angle; the needle should graze the lower end of the body of the sternum and pass up and in, behind the sternum, to the cavity of the pericardium.

Another site often chosen is the fifth left interspace close to the sternum; the needle should pass inward, but there is some danger of piercing the pleura.



**MALIGNANT ENDOCARDITIS THE RESULT OF PYOGENIC INFECTIONS.**

**Etiology.**—The pyogenic micro-organisms are also a cause of malignant endocarditis. Yet it is remarkable, when the frequency of suppurative lesions in childhood is remembered, how very rarely this condition arises.

Osteomyelitis, pneumonia, an abscess in the lung or an abscess resulting from injury may be the starting point of the infection, but the most important group is that which occasionally follows suppuration in the middle ear. This group is one of special interest because the symptoms that arise may very closely resemble those of acute rheumatism.

FIG. 159



Septic endocarditis. (Adams, Jacobi's Festschrift.)

**Symptomatology.**—The symptoms of the malignant endocarditis which results from pyogenic infections are usually more acute and severe than those which follow the rheumatic infection, but the general resemblance is a close one. (See Chap. XXVIII.)

They may be grouped under two headings, viz., those which are the result of the toxemia: irregular fever, sweating, rigors, drowsiness or delirium, diarrhea, progressive anemia, emaciation and purpura; and those which result from the valvular disease: cardiac excitement, precordial pain, valvular bruits, and dilatation (Fig. 159). Fragments of the vegetations detached from the valves will produce infarctions, which

later may give rise to abscesses in the kidneys, spleen, lungs, brain, or even the cardiac wall itself. An aneurysm of the heart may result from severe myocarditis with suppuration. It is sometimes possible to detect this aneurysm by a local bulging of the precordium, or by the development of a bruit, the maximum intensity of which is in some unusual situation, or by a curious whizzing sensation imparted to the hand placed over the heart.

**Prognosis and Treatment.**—These cases of malignant endocarditis which follow pyogenic infections are most fatal, and although preparations of quinine or serum injections are freely used, no method of treatment has met with continued success.

### HEART DISEASE RESULTING FROM SCARLET FEVER.

Heart disease sometimes follows an attack of scarlet fever, as do arthritis and chorea. The result may be a severe and even fatal pericarditis or chronic valvular disease, or, in some rare cases, ulcerative endocarditis. The clinical course of these cases resembles rheumatic heart disease so closely that they will need no detailed description.

The following summaries give an idea of cases which are rapidly fatal:

**CASE I.**—A girl, aged four years, twelve days after the appearance of the rash, developed pneumonia and pericarditis, which proved fatal in a week.

**CASE II.**—A boy, aged six years, six weeks after the rash developed pleurisy and pericarditis and died in a fortnight.

**CASE III.**—A boy, aged seven years, eighteen days after the rash developed pleurisy and pericarditis, which proved fatal in twelve days.

Nephritis may complicate the pericarditis, while in other fatal cases the immediate cause of death has been nephritis, but early endocarditis of the aortic or mitral valve has been discovered at the necropsy.

**Symptomatology.**—The chronic valvular disease following scarlet fever seems to me to be accompanied by more hypertrophy of the heart and more definite symptoms of cardiac distress than that following rheumatism. Certainly, in other respects, this form of heart disease is exceedingly like the rheumatic form, and there is also with it a liability to attacks of acute arthritis.

**Diagnosis.**—This is usually plain, for either immediately after an attack of the scarlet fever there has been a severe cardiac inflammation, or closely following the illness there have been complaints of precordial pain and breathlessness. Again, chorea and arthritis may have occurred during a delay in the convalescence from scarlet fever, and these will suggest the origin of the heart disease.

**HEART DISEASE RESULTING FROM TUBERCULOSIS.**

In tuberculosis of the heart, as in rheumatic disease, the valves and pericardium are liable to damage. It is not at all common in England, at least as a cause of heart disease, although in the postmortem records of the Hospital for Sick Children, Great Ormond Street, there are a considerable number of cases in which tubercles have been observed in the heart wall; in the great majority of these there were no recognizable signs of heart disease during life, and the occurrence was only an incident in a tuberculosis which was more or less generalized.

The following are the chief types: 1. Chronic endocarditis. 2. Pericarditis with or without extensive effusion. 3. Multiple serositis. 4. Malignant endocarditis in a child suffering from tuberculosis.

FIG. 160



**Tuberculous endocarditis.** The left ventricle is opened and shows a large vegetation on the mitral valve. (From the Museum of the Children's Hospital, Great Ormond Street.)

**Chronic Endocarditis.**—In this group occur cases in which the valves only are attacked, and in which calcification sometimes converts the valves and valve-rings into a rigid wall.

There is no history of rheumatism. The occurrence of tuberculosis in other viscera and the dating of the cardiac disorder from an outbreak of tuberculous infection indicate the true nature of the infection (Fig. 160).

They appear clinically as cases of mitral incompetence or stenosis, or, as in one case which came under my notice, of a mitral and tricuspid stenosis. Their course may be very chronic, but there is always the danger of death occurring from some tuberculous affection, and meningitis is especially to be feared.

**Tuberculous Pericarditis.**—Riesman has pointed out the importance of extension of tuberculous disease to the pericardium from caseating



lymph nodes in the anterior mediastinum. The pericarditis is usually chronic and results in dense adhesions, but may be acute with great effusion needing paracentesis. Only in exceptional cases has it been possible to prove experimentally the true nature of the infection. The diagnosis is necessarily difficult, but there is no history of rheumatism, and there may be no valvular disease. These two facts alone, in a child of tuberculous stock, or, still more, in one suffering from tubercle in some other organs, are suspicious. Pericardial friction may be heard in some cases, but in others the acute stage has been so unobtrusive as to be overlooked.

**Tuberculous Multiple Serositis.**—This third group is not sharply differentiated, for valvular damage may occur also. The predominant feature is the occurrence of *pericarditis with pleurisy or peritonitis* or with both. The following case is a good example of the type:

A delicate boy, aged three and one-half years, was said to have had a fit in April followed by diarrhea and some bronchitis. In May he was under observation for feverishness and wasting. There was no personal or family history of rheumatism. In May he also had a definite attack of pericarditis with pericardial friction from which he slowly and incompletely recovered. In August ascites developed, and for this he was tapped on more than one occasion between August and December, and a large and smooth liver was then felt. At the end of December the precordial region was noticed to be prominent and there was exaggerated systolic pulsation with systolic recession of the intercostal spaces. The area of cardiac dulness was enlarged and the heart sounds found muffled, but there was no bruit. Absence of fever had been a feature of his illness, but at the end of December the temperature rose each morning to 100° to 101° F. At the end of January he became drowsy and vomited, then he became unconscious and cyanosed. There was a slight hemiplegia of the right side and the optic disks were blurred. Slight facial twitching was noted later, and he died suddenly, comatose, on January 27th.

The necropsy showed tuberculous meningitis, tuberculous ulcers in the intestine, and caseating bronchial lymph nodes. The pericardium was densely adherent, the heart not noticeably enlarged and the valves normal. There was plastic peritonitis, and around the liver a firm inflammatory capsule. Except for some adhesions the lungs and pleurae were natural.

Another case under my observation, with a history of ascites, ended in the most puzzling manner with tuberculous meningitis, but, in addition, the mitral valve and mitral ring were rigid with calcareous deposits. Some writers consider tuberculosis the most important factor in the causation of multiple serous inflammations.

**Malignant Endocarditis.**—Malignant endocarditis is an occasional incident in tuberculosis, and is probably the result of secondary infection of the valves from some suppurating focus in the lungs or bronchial lymph nodes.



**HEART DISEASE RESULTING FROM INFLUENZA.**

The damage to the myocardium which results from some of the epidemics of influenza is more often seen in the elderly; yet it occurs also in childhood, and even in infancy, as has been described by Forchheimer in Jacobi's *Festschrift*.

**Symptomatology.**—The symptoms are essentially those of acute cardiac dilatation, followed by a more or less prolonged stage of myocardial weakness. Among the symptoms which occur in acute cases, Forchheimer lays stress upon the rapid breathing, resembling that seen in acute edema of the lungs. I would also emphasize the great nervous depression, sometimes quite out of proportion to the severity of the cardiac lesion. The pulse is rapid and irregular and low in tension. As a rule, there is no bruit, but there is dilatation, with feeble cardiac sounds. I am indebted to Dr. Cheadle for calling my attention to cases of influenzal heart disease in childhood, in which there develops a rasping, basal systolic murmur, curiously superficial and clearly audible over the sternum at the level of the aortic cartilage. Whether this is of valvular or pericardial origin is uncertain. These children show symptoms of myocardial weakness lasting, sometimes, for years after the attack of influenza.

Endocardial and pericardial affections are rare, but Austin and others have recorded examples of them.

**Diagnosis.**—Unless there is a history of an attack of influenza, this is not easy. The condition is liable to be mistaken for rheumatism, for there are obscure pains, a sore throat, and dilatation of the heart. The abrupt onset, high fever, nervous prostration, and absence of arthritis and valvular disease are suggestive of influenza.

**Prognosis.**—The prognosis is, on the whole, good, but the weakness of the heart, even at this age, may be very persistent and resist treatment for some years. When infants are attacked the outlook is grave.

**Treatment.**—It is highly necessary in such cases to insist upon rest, immediate and complete. The dilatation should be treated upon the lines indicated under Diphtheria (*q. v.*). Later, athletic exercises will have to be curtailed if there remain shortness of breath, palpitation, and irregularity of the action of the heart.

**HEART DISEASE RESULTING FROM CONGENITAL SYPHILIS.**

It is very doubtful whether congenital syphilis takes anything but a very secondary place in the heart disease of childhood. Gummata and myocarditis have been noted in severe cases of congenital syphilis, but these are pathological curiosities. There are, it is true, some who believe a considerable number of obscure cases of endocarditis are due to this disease, but, for my part, I am doubtful of this and have been struck with the absence of cardiac affections in those infants and children who have shown conclusive evidence of congenital syphilis.

**FUNCTIONAL DISORDERS.**

This is an ill-defined group, for, when one remembers the lesson of diphtheria, one hesitates to apply the term functional to those cases of palpitation, irregularity, and disturbed action of the heart which sometimes follow infectious diseases. The most definite examples are those which result from dyspepsia, with dilatation of the stomach and constipation, and they are especially apt to occur in the children of nervous and dyspeptic parents. In these cases the pulse is irregular and the heart easily excited. Complaints of palpitation and pain are not so usual as a general listlessness and breathlessness on slight exertion, but such children have attacks in which the face is flushed and the action of the heart irregular, rapid, and tumultuous. The cardiac impulse is more than usually visible, although the cardiac area may be very slightly or not at all increased. There is, as a rule, no murmur, although it is not uncommon to meet with a faint systolic murmur, which is audible at the horizontal level of the nipple, and internal to it. Da Costa has directed attention to certain idiopathic cases of cardiac irregularity which appear to run in families, and which apparently improve as adult life is reached.

Functional bruits are not so common in childhood as at and after puberty, but they are met with in anemic children and in anemic, rickety infants. Deformity of the chest resulting from rickets, or spinal curies, or from obstructions in the upper air passages will also give rise to cardiac murmurs; and, if the upper lobe of the left lung is retracted, such murmurs in the pulmonary region may be loud and rasping.

**Diagnosis.**—It is often difficult to decide whether a bruit is functional or organic. A wide survey of the case must be taken, the heart examined most carefully for evidence of hypertrophy, a history of rheumatism inquired for, and cyanosis or slight clubbing of the extremities searched for; it is often necessary to see the case more than once before an opinion that is of any value can be given.

These functional bruits are often modified by the position of the patient and by the respiratory movements, but neither of these facts is conclusive proof of their functional nature.

It is well to acquaint the parents with the fact that there is some weakness of the heart of a passing nature.

**Treatment.**—The treatment is usually satisfactory. Indigestion, constipation, and anemia are corrected. Quiet regular habits and plain meals are necessary, and late hours should not be permitted.

When the digestion is improved, mild tonics, usually prescribed with a saline aperient, are beneficial. The town-bred, nervous caricature of a child will derive much benefit from running wild in the country, but this prescription needs care. Such children will not digest the rough food often met with in farm-houses, and the physician will be greatly blamed if this life is thought by the parents to be too rough for their delicate child.

**ATHLETICS IN HEART DISEASE.**—In this connection it may be serviceable to write a few words upon the subject of athletics. If one can judge by the regulations that are sometimes made, I do not think that a knowledge of heart disease implies any knowledge of athletic pursuits. Possibly, it may seem a trivial matter to be writing upon cricket and football in a solemn work such as this, but the questions that arise are neither easy nor unimportant. Schoolmasters of wide experience will point out that it is very detrimental to interfere unnecessarily with a boy's athletics, and speak of the evils that may result as far outweighing the danger that it is sought to avoid. Boy is a pitiless production, and cannot understand delicacy and feebleness. It is, I think, as a general rule, a mistake to send a boy who has a damaged heart to a big school where athletics are compulsory. But there are many cases on the border-line; these are the troublesome ones, and it is then that a practical acquaintance with the various games is useful.

All *competitive* exercises are dangerous to feeble hearts—I mean by this foot-racing, boat-racing, cycle-racing, cross-country runs, boxing, and so on—for when there is competition the "thorough-bred" will try to better his best. Here lies the mischief, and irreparable damage may be done in this way to a heart which would benefit from ordinary exertion. Acute dilatation of the heart may result from girls dancing too frequently and for too long periods of time.

Football is also dangerous because of the *sudden* exertion and strain inseparable from it. Cricket is more suitable, baseball less so. Fives and lawn-tennis are well enough, if the boy is not permitted to enter for competitive struggles for cups and other trophies. Racquets, if played at all well, is a trying game. Golf is a valuable open-air amusement. Aguin, drilling and graduated gymnastic exercises, though hardly exhilarating, are useful, and may prepare the way for the more active games.

#### THE HEART IN RENAL DISEASE.

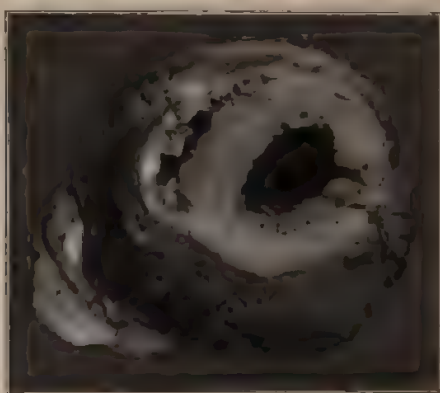
Apart from endocarditis and pericarditis complicating nephritis, renal disease throws a great strain upon the heart.

The first result is, as a rule, dilatation of the left ventricle, and this dilatation may be slight or severe.

Examination will show that the pulse is quickened, low in tension, and sometimes irregular. The impulse is diffuse, the area of cardiac dulness is increased to the left, and the first sound is altered in character. At first there is prolongation, and later shortening of its duration. The rhythm of the heart becomes tic-tac, and both sounds may be reduplicated. Eclampsia is liable to supervene when the heart is dilated and the tension low. Later, hypertrophy of the left ventricle develops (Fig. 161), and the character of the pulse alters; the wave is now prolonged and not easily compressible, and the arterial wall is slightly thickened. It is only in very exceptional cases that advanced arterio-capillary fibrosis and hypertrophy of the heart are met with in childhood.

**Treatment.**—Treatment consists in arresting, if possible, the disease. Rest is essential when there is dilatation. Although there is very rarely any anxiety of a bloodvessel giving way from high arterial tension, still, the tension of the pulse should be kept at a judicious measure by an occasional dose of calomel and saline and by the restriction of meat in the diet. The anemia should be corrected by giving iron combined with a saline aperient.

FIG. 161



Cardiac hypertrophy. A section across the ventricles, showing great hypertrophy of the left ventricle, the result of chronic nephritis in a child. (From the Museum of the Hospital for Sick Children, Great Ormond Street.)

## DISEASES OF THE ARTERIES.

### ANEURYSM.

Diseases of the arteries are rare in childhood, aneurysm being one of the most important. Each case of aneurysm that I have seen myself has been of a different type. One was traumatic in origin, another was the result of malignant endocarditis, and a third was of doubtful nature.

The traumatic case was that of a boy who fell on his head and damaged the right internal carotid artery as it entered the cranial canal. The aneurysm leaked into the throat and the child died after repeated hematemesis.

The case due to malignant endocarditis was a very striking one. The boy had been the victim of severe rheumatic carditis on more than one occasion, and was now under treatment because of a swelling in the right thigh, over the course of the femoral artery. This was clearly an aneurysm, and there was evidence of infarction in other organs; this aneurysm reached an enormous size, and caused terrible pain from the tension in the surrounding tissues.

Death occurred from cardiac failure, and the aneurysm which had



originated from the common femoral artery was discovered to have formed a false sac which occupied all the upper parts of the thigh.

The third case noted in the *Transactions of the Pathological Society of London*, vol. xlvii. p. 24, by Mr. Jackson Clarke, was a girl, aged ten years, who was under the care of Dr. Lees at St. Mary's Hospital. This child had an aneurysm in the left axilla and another in the right buttock. The axillary aneurysm leaked and the child died suddenly. The necropsy showed multiple aneurysms upon the primary and secondary branches of the coronary arteries; there was also a thickened mitral valve, but no recent disease. Congenital syphilis was suggested as a possible explanation.

Jacobi, Sanne, Parker, Keen, and others have recorded cases of aneurysm which serve to remind us that even the arteries, which are the most trustworthy structures in childhood, may sometimes fail. There may be atheroma, or malignant endocarditis, or, as Eppinger's case would seem to show, a congenital lack of elastic tissue. Again, small pulmonary aneurysms are sometimes met with in tuberculous disease of the lungs with cavity formations.

#### OTHER ARTERIAL DISEASES.

**Acute Arteritis.**—Acute arteritis has also been described, and by French writers a good deal has been written upon its occurrence in acute rheumatism. Rabé gives a detailed account (*La presse médicale*, 1902) of this process in the intrapericardial arteries.

I have never met with a conclusive case of acute arteritis in a child, though *perivascular fibrosis* is common in the regions of rheumatic lesions, as is the case in other infections. In malignant endocarditis the inflammation may spread to the commencement of the aorta, and small, white patches of inflammation may also be sometimes seen in simple rheumatism. These differ from somewhat similar fatty patches which are occasionally noticed in necropsies upon anemic children.

ACUTE SEPTIC ARTERITIS sometimes occurs in pyemia.

**General Arteriosclerosis.**—General arteriosclerosis is also rare, but in those unusual cases of granular kidney in childhood it may reach a high degree, and the retinal vessels may then show all the changes which are so well recognized in the disease in adults.

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## SECTION IX.

# DISEASES OF THE GENITOURINARY SYSTEM.

By CHARLES G. JENNINGS, M.D.

### CHAPTER XXXI.

URETHRITIS—VULVOVAGINITIS—DISEASES OF THE BLADDER—  
DISEASES OF THE KIDNEYS.

#### URETHRITIS IN THE MALE.

INFECTION of the urethra is occasionally seen in young boys, and is rarely in male infants. Infection may be by the organisms of pus, *simple urethritis*, or by the gonococcus, *gonorrheal urethritis*.

**Simple Urethritis.**—Pus organisms may invade the urethra from an infected prepuce. Phimosis and uncleanness are the chief etiological factors. A balanitis usually precedes the urethritis. Pus organisms rarely invade the deep urethra; so the inflammation is, as a rule, confined to the fossa navicularis or the first part of the anterior urethra. The prepuce is tender, swollen, and red, and the preputial canal and meatus are bathed in pus. Some pain on micturition is usually present. Retraction of the prepuce is impossible. With cleanliness and appropriate treatment a simple urethritis promptly subsides. It is obstinate when a balanitis and a tight prepuce complicate it. Thorough cleanliness is, under these circumstances, difficult, and often can be attained only by slitting up the prepuce. Careful irrigation of the preputial opening several times daily with a mild antiseptic solution, such as one of weak boric acid, and the removal of all irritating secretions, are the important therapeutic indications. In simple urethritis, injections into the urethral canal are rarely necessary. The administration of alkalies is all that is usually required. Occasionally santal, salol, or oil of wintergreen internally will be needed to control the pathological process in the urethra.

**Gonorrheal Urethritis.**—Gonorrheal urethritis is not infrequently met among the children of the poor and uncleanly. Infants are rarely affected. Boys over the age of six years are the most frequent subjects. Infection may be by venereal contact with an infected member of the family. Pederasty is occasionally the means of infection.

## DISEASES OF THE GENITOURINARY SYSTEM

The demonstration of the gonococcus in the urethral discharge is only certain method of diagnosis of a specific form of urethritis. guide prognosis and therapeutic, microscopic examination of the discharge should always be made.

**Symptomatology.**—Symptoms of a specific urethritis are usually more severe than in the simple form. Pain and swelling often are very marked. The discharge is abundant and composed of thick, creamy pus. A long, tight prepuce is an embarrassing complication, rendering the necessary cleanliness almost impossible and aggravating the urethral inflammation. The gonorrhea of boys is frequently complicated by epididymitis and prostaticitis; cystitis is not so common as in the adult. Orchitis, gleet, and stricture are very rare. The last complication may, however, be overlooked owing to the age of the patient.

In boys, constitutional symptoms are not so severe as in men, and invasion of the joints, the heart, and other remote organs is almost unknown. The carelessness of the child rather favors conjunctivitis. **Treatment.**—The treatment of gonorrhea in a boy is practically the same as in the adult. Cleanliness and the protection of remote mucous membranes by a proper dressing of the parts are imperative. A tight prepuce and a balanitis may demand frequent preputial irrigation and perhaps, operation, although circumcision during the height of a balanitis is not often wise. Urethral injections are not, as a rule, practicable, and in ordinary cases need not be made. They cannot, of course, be entrusted to the patient. In any case, they had better be postponed until the stage of decline. In the acute stage, rest in bed and a properly regulated simple milk and light diet, abundance of fluid, and the administration of alkalies constitute an important part of the treatment. After a few days, salol, 0.3 c.c. (5 min.), every three hours; oil of wintergreen, 0.13 c.c. to 0.3 c.c. (2 to 5 min.), three times daily, or salol, 0.3 gm. (5 gr.), every three hours may be given. Infection of the deep urethra and other complications demand the same treatment as in adult life.

## VULVOVAGINITIS.

According to its etiology, Vulvovaginitis is divided into simple catarrhal vulvovaginitis and gonorrheal vulvovaginitis. **Simple Catarrhal Vulvovaginitis.**—Simple catarrhal vulvovaginitis occurs most frequently in girls between the ages of three and six years. It is frequently seen even in early infancy. Children of hygienic households, those suffering from malnutrition and anemia, are the most frequently affected. It occurs frequently as a complicating local infection in the exanthemata, particularly measles and scarlet fever. The disease is very common where children are segregated, and often occurs in the case of infected nurses, articles of



or instruments. In common with catarrhs of other mucous membranes, it may result from exposure to cold and wet, especially in vitiated states of the system. Traumatism, masturbation, foreign bodies or parasites in the vagina or urethra are occasional causes.

**Pathology.**—The disease is the result of the invasion of the mucous membrane of the vulva or vagina and urethra by one or more of the various pus-producing organisms. The colon bacillus is a frequent offender. The mucous membranes of the healthy and cleanly child resist infection. Diminution of the resistance by one of the above-mentioned predisposing causes is usually a necessary antecedent to the development of the disease.

**Symptomatology.**—Simple vulvovaginitis, as stated above, is most frequently seen in anemic, debilitated children. In such subjects it appears as a mild subacute catarrh with a vulvovaginal discharge that is white or yellowish white. There is some redness and swelling. The vulva is, particularly, the seat of the pathological process, and the inflammation frequently extends to the skin over the vulva and between the thighs. There are no constitutional symptoms. Pain on micturition in the subacute variety is not usually present. Older children sometimes complain of soreness on walking.

In the acute and more severe cases all the symptoms of inflammation are increased. The discharge is a thick, yellowish pus that can be demonstrated to come from the vulva, vagina, urethra, and the cervix uteri. Excoriations of the mucous membrane and superficial ulcerations are common. The discharge forms crusts at the orifice of the vulva, and the labia are adherent. The parts are red, swollen, and edematous, and there is often much local discomfort.

A simple vulvovaginitis usually remains a local process. Remote infections are rare. The communicating lymph nodes are occasionally swollen and tender, but rarely an abscess forms. In colon bacillus infection, particularly, cystitis may result. I have seen one case in which a fatal termination followed successive infection by this organism of the vulva, bladder, and pelvis of the kidney.

**Gonorrheal Vulvovaginitis.**—Bacteriological investigation of vulvovaginitis by many observers has shown that a large percentage of all the cases of the disease are the result of gonococcic infection. Among the negroes of the South, in institutions, in the tenement districts, and wherever uncleanly children associate intimately, the gonococcus appears to be the most common infecting organism.

As in the adult female, gonorrheal infection in the child may pursue a mild or latent course, and numerous observations show that extensive epidemics have been started by infection from children suffering from an apparently trivial vulvovaginal catarrh. As Huber has pointed out, it is often almost impossible to trace the source of an institution epidemic. Transmission of the disease rarely takes place by venereal contact; infection is usually indirect, conveyed by towels, bed-linen, instruments, by nurses, from parents, by contact with soiled floors, etc. Ninety per cent. of the mothers of 44 cases studied by Pott were,

found to be suffering from leucorrhea. Where it has been definitely traced, the period of incubation is about three days.

**Symptomatology.**—While, as a rule, gonococcal vulvovaginitis pursues a more active course than a vaginitis due to a simple infection, there is nothing uniformly characteristic in its clinical history. In the more severe cases there are the evidences of a severe inflammation of all the mucous membranes of the lower genitourinary tract. The vulva and vagina are bathed with a thick, purulent secretion. Holt states that the mucous membrane of the cervix is almost invariably involved. Urethritis is frequent, but does not give rise to such marked symptoms as in the adult. The labia are swollen and glued together with secretion, and the inflammation extends for some distance over the neighboring skin. The inguinal lymph nodes are frequently enlarged and tender; rarely a suppurating bubo results. The glands of Bartholini are often enlarged and occasionally suppurate. During the first few days of severe cases there may be slight fever and constitutional symptoms, although, as Holt remarks, the absence of constitutional symptoms is one of the most striking points of difference between gonorrhea in the child and in the adult.

The course and duration of gonorrheal vulvovaginitis is exceedingly variable. Under favorable conditions complete recovery may take place in from four to six weeks. As in the adult, a specific vaginitis may persist for months, and as a so-called latent gonorrhea the child may be a source of infection for an indefinite period. Relapses after apparent recovery are frequent.

It appears that children resist more vigorously than adults remote gonococcal infection. The literature of the last few years, however, makes it certain that these infections are much more frequent than was formerly supposed, and that the remote effects of a gonorrheal vulvovaginitis are often of the gravest character. In addition to the invasion of the neighboring lymph nodes, the infection may involve the whole length of both the genital and the urinary tracts. Cystitis, pyelitis, and pyelonephritis are occasional results. A frequent result of a long-standing vulvovaginitis is atresia of the vagina. Bokai has reported 39 such cases, and Jacobi mentions it as frequently observed. In atresia from this cause the adhesions are superficial and easily separated by the finger. In all severe cases the endometrium is invaded. Salpingitis, oöphoritis, and peritonitis have been noted, and there may result any of the pelvic complications so frequently observed in adult life—a very important matter when considered in relation to the development of the diseased organs and to future pregnancies.

Gonococcal peritonitis, from infection through the Fallopian tubes, is not uncommon; 40 cases of this character have been collected from current literature. The frequency of this form of infection of the peritoneum should put the physician on his guard in every case of general peritonitis in a young girl. The possibility of confusing this condition with an appendicitis is manifest. The recognition of this condition is of particular importance from a therapeutic point of view.

as the prognosis is so favorable under laparotomy and peritoneal irrigation.

Huber calls attention to a gonococcic proctitis as a not infrequent complication of vulvovaginitis, and he looks upon a lingering infection of the rectum as one of the frequent sources of contagion.

Arthritis as a complication of gonorrheal vulvovaginitis is not common. Koplik has met with three cases; Acker has reported one, in a child two years of age. The possibility of infection of the conjunctiva, although it does not frequently occur, should always be borne in mind.

**Diagnosis.**—The demonstration of the gonococcus in the discharge is the only certain means of differentiating a simple from a specific vulvovaginitis. In the interests of the patient and her associates, this examination should always be made. In the absence of bacteriological demonstration, all severe cases of vulvovaginitis should be looked upon as probably due to gonococcus infection. When several cases occur in a family, in a neighborhood, or in an institution, the chances are strongly in favor of gonococcic origin. The presence of a urethritis, invasion of the urethra, the glands of Bartholini and the upper genitourinary tract point to gonococcic infection.

**Prognosis.**—Simple vulvovaginal catarrh pursues a much shorter and more benign course than when due to gonococcic infection. With judicious treatment recovery will take place in from two to four weeks, although often the case is prolonged on account of the difficulties encountered in the application of local medication. In simple vulvovaginitis complications are rare and are not apt to be of a serious nature.

In gonorrheal vulvovaginitis the progress is much less favorable. Under the most careful treatment cases are obstinate and often are prolonged over a period of weeks or months. Serious and even fatal complications not infrequently occur. The remote effects upon the health of the patient may be unfortunate.

**Treatment.**—As the disease is spread by contagion, prophylaxis is of the greatest importance. In institutions and in families isolation of the patient is essential, and the most scrupulous care is necessary to prevent the spread of the disease. The experience of Huber and others shows how difficult it is in institutions to control the spread of the disease when it has once gained a foothold. Napkins, sheets, towels, and all utensils should be thoroughly sterilized.

For a *simple vulvovaginitis* local treatment consists in absolute cleanliness and the use of mild astringent injections. Twice or three times a day the child should be placed upon a surgical pad or rubber sheet, and the buttocks, labia, and all external parts thoroughly bathed with soap and water and irrigated with a 1:5000 solution of corrosive sublimate or a 1:100 solution of carbolic acid. An antiseptic sitz-bath accomplishes an excellent purpose. Following the external cleansing, an injection from a fountain syringe of from one to two pints of a solution of boric acid, 1:500, or carbolic acid, 1:200, may be used. After the injection the parts should be thoroughly dried and anointed



with cold cream or vaselin. A pad of sterile gauze should be placed over the vulva and held in position by a napkin. With the decline of the acute symptoms astringent injections may be used; tannin or alum solution, 5 per cent.; sulphate of zinc or nitrate of silver, 1 per cent., are efficient.

The general health demands attention, and conditions of malnutrition or anemia should receive appropriate treatment.

In the *gonorrheal* infections the same general plan should be carried out; in the cases in which the inflammation is active the hot sitz-bath will afford great relief. In all severe cases the patient should be kept in bed. Following the cleansing irrigation, the vulva and vagina should be thoroughly douched with a solution of one of the proteid salts of silver; protargol and argyrol are the most efficient. A solution of argyrol, 1 : 200, may be used two or three times a day for the first week or ten days; after this the argyrol may be discontinued and an astringent injection—sulphate of zinc, 1 per cent.—substituted. A surgical wick dressing with a 1 to 2 per cent. ichthyol and a vulvar pad is, in some cases, more efficient than douching. An occasional examination of the secretions for the gonococcus should be made, to determine the progress of the disease. Persistence in the local treatment is necessary for complete recovery. Too early discontinuance very commonly results in a relapse.

#### VESICAL SPASM

Vesical Spasm is a condition quite commonly met with in childhood, and more rarely in infancy. *Dysuria of Childhood* and *Genital Irritation* are synonyms.

**Etiology.**—Vesical spasm may be one of the results of catching cold, or it may occur as a complication of any acute febrile disease. Its most common cause is a highly acid urine and it is most frequently seen when this condition is a result of chronic indigestion with disturbed metabolism. Children of the neurotic type are the most frequent sufferers. It may occur as a complication of vulvovaginitis or urethritis. It is also an occasional symptom of disease of adjacent pelvic organs.

**Symptomatology.**—The chief symptom of vesical spasm is frequent desire to urinate, the act of urination being accompanied by more or less severe pain and vesical tenesmus. The pain attending micturition is often intense and the child, from the great distress, will delay the act as long as possible. Sometimes only a few drops of urine are passed. Usually when once the spasm is relieved the urine passes freely. Examination of the urine, aside from the presence of an excessive acidity, is negative. There is no pus or blood.

The condition may be a passing one, lasting only a few hours or day or, if the cause be persistent, it may continue with exacerbations and intermissions over quite a period of time. In neurotic children with chronic indigestion relapses are common, and any trifling febrile order will again light up the difficulty.



With a careful attention to the diet and the removal of the cause recovery is usually prompt.

**Treatment.**—The child should be given an abundance of a mildly alkaline water. Vichy or one of the lithia waters answers an excellent purpose. Tincture of belladonna or tincture of hyoscyamus, 0.03 c.c. (5 drops), four times a day, may be given. A hot sitz-bath or hot-water applications over the pubes and between the thighs will often give immediate relief to the distress. The diet should be simple and non-stimulating, with milk and cereals in predominance.

### ENURESIS.

In the physiological state evacuation of the bladder follows the natural stimulus of a certain degree of distention of the organ. An afferent impulse passes from the terminal nerves in the bladder to the cord and brain, which send out efferent impulses which contract the detrusor urinæ and inhibit the contraction of the sphincter vesicæ.

In early infancy the evacuation of the bladder is purely a reflex act. At the age of about eighteen months, sooner or later, depending upon the training and also upon the general health of the child, vesical control to a limited degree is acquired. After the third year of life the urine may be held for eight or nine hours during sleep and for two or three hours when awake. Inability to control the bladder after the third year constitutes incontinence.

**Etiology.**—Incontinence is a symptom of numerous malformations and of various organic diseases of the brain and spinal cord. In this article incontinence from these causes will not be considered; they may be studied in the section upon Nervous Diseases.

The ordinary enuresis of childhood is a neurosis. It may have one or more of several etiological factors, viz., elimination of cerebral control over the spinal centres; increase of the irritability of the centres; increase of the irritability of the terminal filaments of the nerves of the bladder or adjacent organs; changes in the composition of the urine.

Persistence of the infantile state, a neurotic inheritance, neurasthenia, anemia, and malnutrition, the debility of convalescence, are conditions in which increased irritability of the spinal centres and of the peripheral nerves is pronounced.

Increased irritability of the terminal filaments of the nerves of the bladder and adjacent organs may be caused by cystitis, urinary calculus, an adherent or tight prepuce, balanitis or vulvovaginitis, rectal polyps, ascarides, or fissure.

A latent chronic cystitis from colon bacillus infection is an occasional cause. In these cases the micro-organism may be demonstrated in the freshly passed urine, which may be normal in appearance or but slightly turbid. The only other symptom may be the enuresis.

A highly acid and irritating urine is often present and sometimes a cause of enuresis. More often it is only an associated phenomenon,

resulting from the same malnutrition factors that determine the irritability of the nervous mechanism of micturition. In many cases the most careful investigation fails to reveal an adequate cause for the condition. Not infrequently nocturnal incontinence is met with in children otherwise apparently in robust health. To this class of cases some writers have confined the term "enuresis."

Incontinence occurs with equal frequency in both sexes, both in private and institution practice. Most cases are seen during the middle period of childhood.

**Symptomatology.**—In enuresis proper there is no dribbling. The bladder, when full, empties itself fully and freely without the intervention of the will. The reflex mechanism responds so promptly to the peripheral irritation that the child, even when awake, may have no power of postponement. The enuresis may be nocturnal or diurnal, or both. Nocturnal incontinence is the more frequent. There are all grades of severity. In some children there is only an occasional lapse under the influence of some distinct cause, while in others the bed is wet every night and even several times a night. The condition may continue to late childhood, and even to puberty. After puberty nocturnal emissions may replace the incontinence.

**Prognosis.**—When incontinence is traceable to a distinct cause that is removable the prognosis is good for prompt relief. While some cases quickly respond to medical treatment, a large number require patience and persistence over a number of months or years. The condition in any case will be more difficult to overcome in proportion to the length of time it has continued.

**Treatment.**—If a cause for the trouble can be found it should be removed. Adherent prepuce, phimosis, narrow meatus, chronic latent cystitis, vesical calculus, hyperacid urine, ascariides, vulvovaginitis, rectal diseases—all should be sought for and, if present, receive proper treatment. Circumcision is a measure usually advised and carried out, but it must be confessed that alone it rarely influences the condition. With a redundant, tight prepuce it is probably an important preliminary treatment. Remote local causes of heightened reflex irritability, such as tonsillar hypertrophy and adenoids, should receive attention.

Treatment of the general condition of the patient is essential. Anemia, malnutrition, constipation, and chronic indigestion should be treated with appropriate tonics, a careful diet and an out-of-door, simple life—free from the mental worry of school competition. Without attention to these points, any medical treatment is unavailing.

The correction of dietetic errors is essential in every case. Sweet and pastry, hot breads, cake, indigestible meats, tea and coffee should be prohibited. An excess of uncooked fruit will often keep up chronic intestinal indigestion. No food should be allowed between the regular meals except a half-glassful of milk. A light supper and not more than one glassful of fluid should be given with it. No liquid should be taken after supper. On rising the child may be given a quick, cold sponge bath followed by a vigorous rub.

The specific treatment for the direct control of the enuresis should begin with tincture of belladonna, 0.06 c.c. (1 drop) to each year of the child's age, increasing the dose by 0.06 c.c. (1 drop) each day until the enuresis is controlled or the physiologic action of the drug is manifest. A dose of 0.6 to 0.72 c.c. (10 to 12 drops) is often necessary. When a controlling dose is reached, it may be held for a week or two and then carefully decreased, increasing it again from time to time, if necessary, to maintain the therapeutic effect.

Should belladonna fail to control the enuresis, a solution of atropine and strychnine containing 0.13 gm. (2 grains) of atropine and 0.065 gm. (1 grain) of strychnine to 30 c.c. (1 ounce) of water may be prescribed. One drop of this solution should be given three times a day, and increased one drop a day after the manner of the administration of the belladonna. The strychnine is particularly valuable in diurnal incontinence. *Rhus aromatica*, 0.60 c.c. to 1.25 c.c. (10 to 20 drops), is often useful either alone or combined with belladonna. In highly nervous children potassium bromide is sometimes a useful addition.

The belladonna treatment should be continued over a period of two or three months or more, if necessary. With the onset of cool weather and following dietetic errors, or mild derangements of health, relapses may occur. Prompt renewal of the treatment will be necessary.

Faradism, with the positive electrode in the rectum and the negative electrode over the pubes, may be tried in obstinate cases, although in my experience it is not often of use. Holt suggests, in old cases with probable contracted bladder, the daily distention of the organ to its normal capacity, with warm normal saline solution, and it is worth a trial.

#### THE URINE.

The studies, especially of Holt, Jacobi, and Morse in this country, and of Baginsky and others in Europe, have revealed the previously unsuspected frequency of diseases of the urinary organs in infancy and childhood. While in the diseases of adult life the examination of the urine is a routine measure with all careful diagnosticians, the real and fancied difficulties in obtaining a specimen of urine from the infant have deprived the physician of this prompt and essential means of diagnosis, and many cases of urinary disease have passed unrecognized. Baginsky believes that many deaths from eclampsia in babies are really caused by uremic convulsions, and it is a common experience with the consultant in diseases of children, to find the diagnosis of a puzzling case made plain by urinary analysis. The importance of urinalysis in cases of scarlet fever, pneumonia, influenza, diphtheria, gastro-enteric catarrh, etc., is not appreciated, and it is unfortunately too often omitted.

To collect a specimen of urine from the male infant, a small open-mouth bottle, with an short neck, or, what is much better, a rubber pouch or condom, may be adjusted over the penis at a reasonable time

following the last micturition. From the female infant, to obtain a specimen is more troublesome. A bottle or pouch may be fixed over the vulva with adhesive plaster in the same manner as in the male. A clean, well-washed sponge placed over the vulva, under the diaper, is an easy and often satisfactory method; or, the baby may be placed, about the time for urination, in its crib on a rubber sheet, under the observation of the nurse. The application of a cold cloth over the region of the bladder will often stimulate micturition. Wherever it is necessary, there should be no hesitancy in passing a catheter. A small, clean, soft-rubber catheter, passed with the well-known precautions against infection, produces only insignificant discomfort and is always harmless. Should a twenty-four-hour specimen be required, this method should always be used.

The urine of the newborn infant is small in amount, rarely more than two to eight ounces being passed in twenty-four hours. Complete anuria for the first twelve to twenty-four hours after birth is not uncommon. While it always should receive careful attention, it is usually of no significance and secretion is established with the administration of an abundance of water. The first urine drawn with the catheter is usually clear, with a specific gravity of 1.006, small in amount and feebly acid. On the second or third day it usually becomes cloudy, strongly acid, highly colored, and with a specific gravity of 1.010 to 1.012. Napkins are stained by the uric acid crystals. The high relative proportion of this constituent of the urine the first few days of life produces the condition known as uric acid infarct of the kidney. The urine is strongly acid. It contains often a large amount of mucus, which may easily be mistaken for albumin. This mucus is probably the result of irritation of the bladder from the highly acid urine. Hyaline casts are not infrequently found and epithelial elements are abundant. The phosphates do not appear until about the fourth day. Subsequently and throughout early childhood the specific gravity of the urine is low (1.004 to 1.008), and the coloring matter and other salts, with the exception of uric acid, relatively small in amount. The percentage of uric acid and urea remains high during childhood.

Albumin and sugar are occasionally present in the urine of otherwise apparently healthy children during the first month or two of life. Sugar may be present in the urine of infants overfed with patented foods.

Published studies are not adequate to permit the compilation of an accurate table of the quantity in twenty-four hours, the specific gravity, and the percentage of the normal constituents of the urine of the healthy child. Great variations are found in the results obtained by different observers, and it is difficult to account for these differences unless it be acknowledged that the urine excretion in infants and children is subject to great unexplained physiological variations. The following table, compiled from the studies of Holt, Churchill, Morse, and other observers, gives an approximate average of the amount in twenty-four hours, the specific gravity, and the urea content of the urine during the first ten years of life:



Age.	Amount in twenty-four hours.	Specific gravity.	Urea.
First week . . . . .	8 to 90 c.c.	1.010 to 1.004	0.07 to 0.66 grams.
Third month . . . . .	200 "	1.004 " 1.010	1.4 " 2.3 "
Sixth " . . . . .	250 "	1.006 " 1.012	5.0 " "
Ninth " . . . . .	300 "	1.006 " 1.012	7.0 " "
First year . . . . .	400 "	1.006 " 1.012	11.0 " "
Second year . . . . .	450 "	1.006 " 1.012	12.0 " "
Third " . . . . .	500 "	1.006 " 1.012	13.0 " "
Fourth " . . . . .	550 "	1.008 " 1.016	13.5 " "
Fifth " . . . . .	600 "	1.008 " 1.016	14.0 " "
Sixth " . . . . .	650 "	1.008 " 1.016	15.0 " "
Seventh " . . . . .	700 "	1.008 " 1.016	16.0 " "
Eighth " . . . . .	800 "	1.008 " 1.016	18.0 " "
Ninth " . . . . .	900 "	1.010 " 1.020	19.0 " "
Tenth " . . . . .	1000 "	1.012 " 1.020	20.0 " "

**Suppression of Urine in the Newborn.**—This condition occasionally results from an acute renal congestion, due to the irritation of uric acid infarcts; it is best described in the following record: Male child, born April 1st, breech presentation. Urinated freely at birth. Suppression of urine occurred on the fifth day and continued six days. During this time, so far as known, no urine was passed. Several warm baths were given during this period and the infant may have urinated then. The temperature ranged from normal to 103° F. Four mild convulsions occurred. There was no dropsy. A specimen of urine obtained April 13th contained albumin in small quantity, many uric acid crystals, red blood cells, granular casts, and a few small, round epithelia. In a few days the urine cleared. On April 24th it contained but a faint trace of albumin and only one hyaline cast was found. On April 16th profuse umbilical hemorrhage occurred and a less severe intestinal bleeding. Many purpuric spots showed on the body and one very large one on the chest.

Complete recovery followed the disappearance of the purpura.

#### CYSTITIS.

Escherich, Jacobi, and other writers in recent years have insisted on the comparative frequency of cystitis in infancy and childhood. In infancy it most frequently occurs as a complication of simple or specific vulvovaginitis or enteritis. Often the three diseases coexist. Infection in these cases takes place by way of the urethra or through the lymph channels from the intestine. The colon bacillus is the most frequent infecting organism. Other infections that have been reported are the bacillus proteus, the bacillus pyocyaneus, and the various cocci. Tuberculous infection is not unusual in the general tuberculosis of childhood. Infection may take place from a neighboring abscess, as in the perineum, or from a diphtheria of the vulva. Foreign bodies, traumatism, calculus, a highly acid urine, and exposure to cold are among the predisposing causes of infection. Any obstruction to the escape of the urine predisposes to cystitis.

**Symptomatology.**—Cystitis is not infrequently discovered in children under treatment for other diseases. Careful inquiry in these cases elicits a history of mild vesical symptoms. Such mild cases complicating other diseases usually promptly recover without special treatment, and it is not improbable that many of them run their course unrecognized.

In the more pronounced cases there is acute pain in the perineum and region of the bladder; there is tenderness on pressure over the pubes, and extreme irritability of the bladder with frequent micturition, which is accompanied by great pain and tenesmus, especially toward the end of the act. In severe cases convulsions may occur. Fever and constitutional symptoms are present in all severe cases.

The urine, which is passed in small quantities, is highly colored, cloudy, and neutral or alkaline in reaction. It contains considerable mucus and a small amount of albumin, due to pus. Microscopically there are found pus, blood cells, proliferating epithelial cells, and great numbers of bacteria. In hemorrhagic cystitis the urine is bright red from the contained blood, and often contains small clots. In the later stages a bad-smelling, alkaline urine with abundant deposit of phosphates is characteristic.

In chronic cystitis the same general symptoms are present, although the bladder is less irritable and the distress more bearable. With boys itching or pain at the end of the penis is frequent.

A latent chronic cystitis may result from an acute attack and persist unrecognized for an indefinite period. In these cases the colon bacillus may be demonstrated in the freshly voided urine, although on inspection it may appear normal or but slightly turbid. This latent form of cystitis is most frequent in girls and may be the cause of an otherwise unaccountable debility or enuresis.

**Diagnosis.**—Cystitis may occur in conjunction with almost any disease of the genitourinary tract. Its special diagnostic features are the frequent and painful urination and the composition of the urine. The presence in the urine of considerable amounts of mucus, pus, and bladder epithelium, with a relatively small amount of albumin in the filtered urine, all speak for cystitis, and these qualities together with the absence of casts exclude nephritis. Pyelitis may lead to vesical irritability. There is, however, in pyelitis no tenderness of the bladder on bimanual palpation and there is tenderness in the kidney region. The continued fever of pyelitis is not present in cystitis. Bacteriologic examination for the definite recognition of the infecting agent should be made whenever possible.

**Prognosis.**—The marked tendency of the mild cases to spontaneous cure has already been referred to. Under treatment the simple case recover in ten days or two weeks. Gonorrheal cases will be obstinate. The latent form may run on indefinitely unless recognized and properly treated.

**Treatment.**—In mild cases rest in bed and demulcent drinks may be all that is required. In the severe cases additional treatment will be necessary. Hot sitz-baths and the application of hot-water fomenta-

tions over the pubes and perineum are valuable. In the early stage the use of hyoscyamus with alkalies gives prompt relief. For severe pain an opium suppository may be used or syrup of Dover's powder administered. The diet should be milk and the cereal gruels. Bladder irrigation is difficult in children and is not often necessary. When pus is abundant and the urine alkaline, urinary antiseptics, urotropin, benzoic acid, boric acid, or sandalwood oil may be given. When vesical irrigation is necessary, solution of boric acid, 0.324 to 0.650 gm. to 30 c.c. (5 to 10 gr. to 1 oz.), or carbolic acid, 1:200, are the most useful. In every case a careful search for calculus or other removable cause should be made.

Chronic cystitis should be treated by the persistent administration of salol and urotropin along with a strict milk diet. Occasional bacteriological examination of the urine will be necessary to determine the progress of the case toward recovery.

#### ALBUMINURIA.

While late investigations, particularly those of Mörner, have proven that minute traces of albumin are present in normal urine, the source of which, whether from the kidney or the lower portion of the urinary tract, is undetermined, the presence of albumin in amount sufficient to be detected by the usual clinical methods, unless due to admixture below the kidneys, must be looked upon as evidence of failure of the renal epithelium to perform its normal function.

**Albuminuria in Early Infancy.**—Albuminuria is an almost constant phenomenon during the first four or five days after birth. In many cases it persists for two or three weeks and, not infrequently, for two months. The cause of this albuminuria is uncertain. Many believe it to be a physiological condition. It has been attributed to circulatory changes at birth; to postnatal readjustment of metabolism; to maternal renal disease; and to lithemia, so constant a condition in the first few days of extrauterine life.

This albuminuria of early infancy is transient and has no prognostic significance. Except in early infancy, albuminuria occurs in early life under the same conditions and has the same significance as in the adult.

**Albuminuria in Later Infancy and Childhood.**—Albuminuria is a characteristic symptom of acute and chronic parenchymatous degeneration of the kidneys, of acute and chronic nephritis, and of amyloid and fatty degeneration. It is often present in renal new-growths, perinephritis, and abscess. It is an associated phenomenon in hematuria and hemoglobinuria and in the various pathological conditions of the genitourinary tract, attended by the formation of pus. A slight albuminuria is often observed in various constitutional conditions: anemia, scurvy, purpura. It is often present in jaundice and glycosuria. It is common after epileptic seizures, and has been found after anesthesia.

According to Rachford, it is frequently present in early childhood in lithemia and other toxic states.

A transient albuminuria is sometimes due to a movable kidney, and in this condition it is particularly noted after exercise. After a vigorous palpation of a movable kidney, albuminuria has been observed. An afternoon albuminuria is a frequent symptom of pelvic calculus.

In most of these conditions the amount of albumin is small; an abundant albuminuria is observed only in grave acute or chronic organic disease of the kidneys.

### FUNCTIONAL ALBUMINURIA.

Functional Albuminuria is characterized by the appearance of albumin in the urine in quantities easily recognized by ordinary clinical methods, continuously or during certain hours of the day, and the absence of other symptoms of organic disease of the kidney. Based upon its supposed etiology or its clinical characters, writers have designated the affection *neurotic*, *dietetic*, *cyclic*, *intermittent*, and *paroxysmal albuminuria*. Under the term *cyclic albuminuria* most of the literature, which is not abundant, has been written.

A study of the literature of this subject shows the utmost confusion in the minds of observers as to what constitutes a cyclical albuminuria. In many of the cases reported, the clinical history and the urinary findings show conclusively the presence of organic renal disease, the only excuse for terming the condition a cyclical albuminuria being the phenomenon of an albuminuria absent or slight during periods of rest and more or less abundant after physical exercise. I believe that the term functional or cyclic albuminuria should be confined strictly to cases in which the clinical phenomena and urinary findings indicative of renal disease are absent.

**Etiology.**—The condition is rare in infancy and early childhood, although not infrequently met with in later childhood and adolescence. It is most frequent in boys. Cold bathing and severe muscular exertion are among the most frequently noted exciting causes. Dukes in the Rugby School found it in many boys subjected to sharp morning exercise. It is occasionally one of the associated phenomena of chronic indigestion and lithemia. A diet too rich in proteids may be the cause.

**Pathology.**—The pathology of cyclical albuminuria is obscure. Renal irritation from lithemia, vasomotor disturbances, and deranged metabolism, with the formation of proteids capable of transudation through the normal kidneys, are among the most reasonable pathological explanations of the disease. An albuminuria the result of degenerative or inflammatory changes in the kidney, however slight or evanescent, is not a functional albuminuria.

**Symptomatology.**—Many patients are well nourished and show no symptoms other than the albuminuria, which may be discovered acci-



dentally. According to Baginsky, these patients are frequently pale, thin, and spiritless. Some have chronic indigestion, are anemic, and suffer from various neuroses.

The amount of albumin in the urine is usually small, although Tyson says that with the heat test it may amount to one-half the bulk of the urine. The albumin may be more or less continuously present, or it may be intermittent. Typical cyclical albuminuria is characterized by a urine free from albumin in the early morning, and containing albumin during the hours from about ten o'clock in the morning until late in the evening. That rest is the important factor in checking the excretion of albumin is shown by the effect of a stay in bed. The albumin disappears, only to reappear on the resumption of exercise. While twenty-four hours is the usual cycle, longer periods are recorded. Dornig cites a case with a Sunday albuminuria. An increase of the phosphates is common, and Holt speaks of an occasional glycosuria. The urine is not diminished in amount, and the specific gravity is normal or high. The sediment frequently contains uric acid, urates, phosphates, or oxalates.

**Diagnosis.**—An albuminuria should be declared functional only after a complete physical examination and repeated urinary analyses have failed to reveal the presence of the clinical and laboratory evidences of organic renal disease. The presence of edema, cardiac hypertrophy, high pulse tension or retinal changes, with or without other evidences of impaired general health, means organic kidney disease regardless of any peculiarity in the course of an albuminuria. Deficient excretion of uræa and the presence in the urine of hyaline and epithelial casts, blood, pus, and renal epithelium have the same significance. Tyson well says that "the most important injunction in the recognition of this form of albuminuria is a careful and exhaustive examination for casts." Even an occasional hyaline cast should be looked upon with suspicion, particularly in the absence of any cause of acute degeneration of the kidney.

**Prognosis.**—The prognosis of a true functional albuminuria is favorable. The condition may pass away in a few weeks or persist for months. Certainty in diagnosis, however, is essential to a favorable prognosis, and every case of albuminuria should be viewed with suspicion until its disappearance. A chronic nephritis in childhood is often insidious and deceitful. The persistence of an albuminuria beyond a few months is strongly suspicious of organic disease of the kidney.

Three cases were observed by me over a period of ten to twenty years. The first was a medical student, and the albuminuria was persistent and abundant during the whole of one winter. There were no other evidences of renal mischief. The patient was under observation during the winter of 1882. Since leaving college he has had no return of the trouble. The second was a hospital nurse, observed during the years of 1890 and 1891. She had a persistent mild albuminuria with no other evidences of renal disease. The albumin disappeared after a few months and she has remained perfectly well since, so far as kidney

disease is concerned. The third case was a young man who had a rather abundant albuminuria and was under observation for a period of two years. This case was carefully studied. In addition to the albuminuria hyaline casts were occasionally found. This condition continued, without disturbance of the general health, for about five years, then characteristic symptoms of chronic nephritis appeared, and the young man died about two years later.

My experience agrees with that of most authorities, that a persistent or an intermittent albuminuria, while it may be present without any other evidences of renal mischief and ultimately pass away, should be looked upon always with concern.

**Treatment.**—No drug with which we are familiar will influence the excretion of albumin in the urine. Treatment must be directed to the disturbances of digestion and metabolism that, in all probability, lie at the foundation of the condition. Complete rest is often essential.

A carefully regulated diet and a healthy out-of-door life, free from excessive muscular exercise, are of the most importance. The diet should be liberal and carefully selected. Excess of proteid food should be avoided. Digestive derangements and anemia should be treated. Iron and arsenic in small doses are valuable. A cool morning bath, followed by a good rubbing, is a valuable vasomotor tonic. Should the condition persist, a winter in a mild climate, away from sudden changes in temperature and high winds, would be advisable.

### HEMATURIA.

Blood in the urine is a symptom of a number of pathological conditions, and it may have its origin from any part of the urinary tract. While usually symptomatic, a number of cases have been observed in which the hematuria was apparently due to an idiopathic renal hemorrhage, a renal epistaxis, as Durante has termed it. Senator designates the condition "renal hemophilia."

Hematuria in early life more frequently has its origin in the kidney. Traumatism of the urethra and bladder, calculus in the bladder, and, rarely, new-growths in the bladder may give rise to this symptom.

Calculus in the ureter or kidney is an occasional cause. Hematuria is one of the most important symptoms of renal sarcoma, occurring in nearly half the cases, and is frequently the first symptom noted. It is very common in active and passive renal congestion and nephritis. The characteristic red or smoky appearance of the urine in the early stages of nephritis is due to the presence of blood. It occasionally is a symptom of the infectious diseases—typhoid fever, virulent scarlet fever, and influenza. Its appearance in these diseases is often indicative of the onset of nephritis. According to Thayer, malaria never produces true hematuria in children. Syphilis is a possible cause. It is a rare manifestation of hemophilia and hemorrhagic disease of the newly born. It has been noted in a number of cases of infantile scurvy, it being the first and

only symptom in a number of cases collected by the American Pediatric Society. I saw one case, in consultation, in an infant six months old. The child had been passing blood for four weeks. No other scurvy symptoms were present. A change of diet promptly relieved the condition. Morse also cites three cases in which hematuria was the only characteristic symptom of scurvy.

**Diagnosis.**—A hematuria having its source in the kidney is often intermittent. The blood is thoroughly mixed with the urine, and, when voided, is equally bloody at the beginning and at the end of micturition. Blood casts of the uriniferous tubules and clots formed in the ureters are characteristic of renal hemorrhage. Pain is the only distinguishing characteristic of ureteral hemorrhage.

Hemorrhage from the bladder is apt to be continuous. The first urine voided is light and contains little blood. Toward the end of micturition the color becomes deeper and pure blood may be passed. Pain and tenesmus are usually concomitant symptoms.

Blood from the prostate and urethra appears in the first part of the discharge, the urine voided last being clear and free from admixture with blood. Pain at the end of micturition is frequent.

The color of urine containing blood varies from a smoky tint to a dark red. The quantity of blood passed in the urine may vary from an amount recognizable only by the microscope to a number of ounces. The passage of large quantities of blood is characteristic of the renal hemorrhage of sarcoma.

**Treatment.**—The treatment of a hematuria will depend upon the cause. Rarely is it of sufficient abundance to demand measures for its arrest. Rest in bed, iron, alum, and adrenalin chloride are the most efficient remedies. Gelatin by the stomach and hypodermically has been used.

### PYELITIS.

Pyelitis is an inflammation of the pelvis of the kidney. When complicated by extension into the tubules of the kidney it is termed *pyelonephritis*. When it results in an accumulation of pus in the pelvis of the kidney it is termed *pyonephrosis*. The disease may be primary or secondary, acute or chronic.

PRIMARY PYELITIS is not a common disease, although cases are met occasionally in infant hospitals and in private practice by physicians who make it a rule to examine the urine of sick infants. Many cases undoubtedly escape recognition because of neglect of urine analysis.

Most of the reported cases have occurred in female infants. The two cases I have seen were in female infants under one year of age.

The colon bacillus is the usual infecting organism, and it may gain entrance to the pelvis of the kidney from the intestinal contents by way of the urethra, bladder, and ureter, or by the blood or lymph channels. It is significant that many cases are preceded or attended by mild intestinal disorders.



SECONDARY PYELITIS is more common than the primary form. It occurs not infrequently secondary to cystitis from colon bacillus or other infection, and more rarely as a complication of gonorrheal vaginitis with or without the intervention of a cystitis. Irritation of the pelvis from renal calculi is a frequent cause. It may result from malformations, renal tuberculosis, renal tumors, perinephritis and perinephritic abscesses, and pyemia, and it may occur as a complication or sequel of several of the acute infections, especially scarlatina, diphtheria, measles, and typhoid fever.

**Symptomatology.**—*Primary pyelitis* usually begins abruptly. The onset may be marked by a chill, which may be repeated at irregular intervals during the course of the disease. In the two cases seen by me, the chills were absent. The temperature rises rapidly, often marking  $105^{\circ}\text{F}$ . or even higher, and is accompanied by the usual symptoms of fever. The course of the fever is irregular. The temperature may continue high, with but slight remissions, or it may show sharp remissions or intermissions.

The remarkable feature of the disease is the absence of local symptoms that would indicate that the pelvis of the kidney is the seat of trouble. In rare cases some evidence of pain and tenderness over the region of the kidney may be elicited. Occasionally a mild intestinal disturbance, as shown by abnormally frequent and changed stool, may precede or accompany the pyelitis. Unless the local disease is recognized and properly treated it may progress for several weeks or longer, with the wasting, prostration, and other symptoms that result from high fever.

Examination of the urine reveals the nature of the trouble. The urine is scanty, acid in reaction, and turbid from the presence of pus. Albumin is present in small amount, corresponding to the amount of pus.

The microscope shows, in addition to the pus, spindle and cauda epithelial cells from the pelvis of the kidney, a few hyaline casts, and often crystals of uric acid. In recent or severe cases red blood cells. The colon bacillus in pure culture may be found.

In *secondary pyelitis* the constitutional symptoms may be obscured by those of the primary disease and the pyelitis may be recognized only by the pyuria. When complicating cystitis there is frequent and often painful micturition. In pyelonephritis a more abundant albuminuria is present, with blood, renal epithelium and hyaline, granular, and epithelial casts. Pyuria, renal colic, hematuria, and pain and tenderness in the region of the kidney, together with fever, are the characteristics of pyelitis complicating renal calculi. Pyelitis may be a symptom of renal tuberculosis. The demonstration of the tubercle bacillus in the urine with the evidences of general infection reveal the nature of the primary disease. Pyelitis secondary to renal tumors, abscess, and perinephritis is usually unilateral and shows characteristic local symptoms. A chronic pyelitis may pursue an afebrile course or be marked from time to time with periods of high temperature.



**Diagnosis.**—Without urinary analysis primary pyelitis may be confused with any of the acute febrile diseases. There are usually no symptoms that attract attention to the urinary organs, and it is usual for these cases to be diagnosticated typhoid fever, malaria, or fever from acute intestinal toxemia. Pyelitis should be suspected in every case of unaccountable fever in infancy. The diagnosis can be made positive only by the microscopic examination of the urine. The presence of pus in an acid urine, together with the chills, high irregular temperature, and perhaps pain and tenderness in the region of the kidney, are characteristic.

With a complicating cystitis there are, in addition, vesical pain, frequent urination, and in the urine numbers of bladder epithelial cells. The possibility of tuberculosis should be kept in mind in every case of pyelitis.

**Prognosis.**—Under proper treatment primary pyelitis usually pursues a favorable course, terminating in complete recovery in from two to four weeks. If unrecognized it may progress indefinitely and death may result from exhaustion or some secondary infection. The prognosis of pyelitis complicating other diseases will depend upon the nature of the primary disease and upon the treatment.

**Treatment.**—At the beginning of the attack the bowels should be well cleared with calomel or castor oil. Subsequently the colon may be flushed with normal saline solution every day or so. The diet of artificially fed infants should be adjusted to the digestive state. An abundance of water to thoroughly flush the kidneys should be given with moderate doses of an alkali to neutralize the excessive acidity of the urine. Citrate of potassium, 0.12 to 0.18 gm. (2 to 3 gr.) well diluted may be given every two hours during the day. Urotropin is the most important remedy for controlling the pyuria. It may be administered to an infant one year old in the dose of from 0.03 to 0.12 gm. ( $\frac{1}{2}$  to 2 gr.) every three hours. The effect of urotropin must be carefully watched, as it sometimes irritates the kidneys and bladder. The efficiency of the remedy as a urinary antiseptic is impaired in a highly alkaline urine, and in some cases with such urine sodium benzoate may be substituted for the potassium citrate with advantage.

The fever and constitutional symptoms are best controlled by hydrotherapy.

In the subacute or chronic stage Jacobi thinks well of gallic acid, 0.6 to 1 gm. (10 to 15 gr.) in the twenty-four hours.

#### ACUTE DEGENERATION OF THE KIDNEYS.

Delafield, Prudden, Holt, and others have clearly brought out the clinical and pathological relations of Acute Degeneration of the Kidney. By many writers and clinicians the disease is confused with acute nephritis. Although recognizing the pathology of the condition, Morse and Kelly have named it "acute degenerative nephritis." Some English writers use the term "nephritis" to designate this condition, reserving

influenza and malaria, and local pus infections. It is one of prolonged high temperature from any cause. It is found in the autopsies on children dying from the acute infection to the bacterial poisons, it may be caused by the various of erroneous metabolism. It is thus found in jaundice and lithemia. The ingestion of irritating and toxic drugs—salicylates, turpentine, arsenic, and phosphorus—may cause it.

**Pathology.**—Acute degeneration of the kidney is the result of the action of various toxic substances upon the renal epithelium, the elimination of these substances through the kidney. Changes in the epithelium of the glomeruli and tubules, and in the tubules, fatty degeneration and necrosis, are the distinctive pathological changes. Congestion and the exudation of serum may accompany it. The kidneys are slightly enlarged, soft, and pale.

**Symptomatology.**—Acute degeneration, as it is usually seen in children during one of the acute infections, runs its course with symptoms additional to those of the primary disease. It can be recognized only by urinary examination.

The characters of a febrile urine are present. The quantity is small and the specific gravity high, 1.022 to 1.030. It is turbid and high colored. Albumin in but a small amount is present. In some cases, particularly in diphtheria, the amount is large. A few hyaline or granular casts, epithelial cells and occasional pus cells are found in the sediment. The urine is normal with the end of the primary infection.

**Diagnosis.**—The presence of the urinary findings of acute degeneration often leads to an erroneous diagnosis of serious renal disease. Acute nephritis is a not infrequent complication of the acute infection. Holt, Morse, and others have shown that its frequency has been exaggerated by many writers, who have accepted the urinary findings of acute degeneration as indicative of the more serious disease.

convalescence the condition usually disappears. In severe infectious diseases it may, however, interfere with renal excretion and contribute to a fatal termination. There is no evidence to show that a kidney the seat of an acute degeneration is rendered more susceptible to acute inflammation later in the course of the primary infection.

**Treatment.**—As a rule, no treatment other than that for the primary disease is required. If excretion be defective, an abundance of fluid and a diet and medication selected with the view of producing the least possible irritation of the kidneys are advisable. In gastroenteric diseases the relief of the irritation by proper diet, etc., will frequently end the symptoms of the degeneration.

### ACUTE NEPHRITIS.

This condition has been also described as *acute exudative nephritis*, *productive nephritis*, *diffuse nephritis*, *glomerulonephritis*, *parenchymatous nephritis*, *catarrhal nephritis*, and *acute Bright's disease*.

**Etiology.**—Acute nephritis may be primary or secondary. Of the two forms, the secondary is by far the more frequent. From the literature and his own experience, Holt collected twenty-four cases of primary nephritis in infants under the age of two years. I have observed but one, a fatal case. In older children, also, the primary form is rare. I have seen three cases in consultation in the last two years, all of them fatal. Exposure to cold and wet is the probable cause of the primary form.

The most frequent cause of secondary nephritis is one of the acute infections, especially scarlet fever and diphtheria. While more frequent in the severe cases of these diseases, it may occur even in the mildest form and regardless of every precaution for its prevention. It is an interesting fact that nephritis is usually a late complication or a sequel of scarlatina.

While the disease is due to the direct toxic action of the scarlatinal virus on the kidney, it would thus appear that the secondary streptococcus infection may also play an important role.

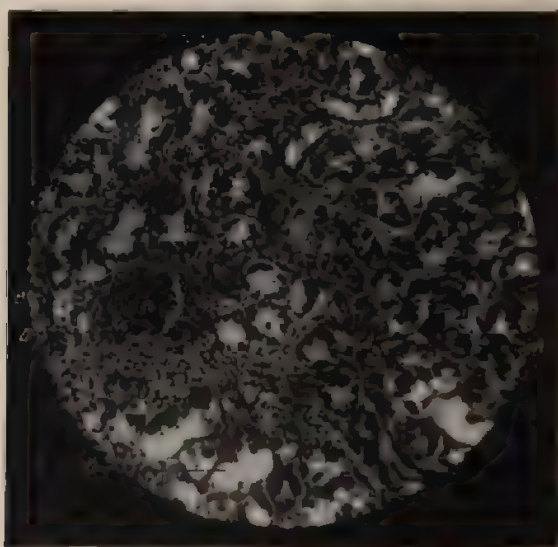
More rarely it complicates measles, epidemic parotitis, varicella, variola, typhoid fever, pneumonia, and meningitis. The literature of the last few years contains numerous reports of cases complicating influenza, malaria and tonsillitis. Considering the great prevalence of influenza during the last fifteen years and the few cases of nephritis reported, it must be looked upon as a rare complication. Personally I have observed but one case. Local and systemic pus infections, erysipelas, dysentery, acute rheumatism, impetigo, and pustular eczema are occasional causes. As in acute degeneration, leucemias and chemical poisons may also produce nephritis. Infants and children of any age may have nephritis, although it is more common in the middle period of childhood. Boys, from their more careless lives, are more frequently affected.



**Pathology and Pathological Anatomy.**—The determination of nephritis as a complication of one of the infections is usually the direct result of the action of a toxin, elaborated by the provoking micro-organism, during the process of its elimination by the kidney. In some of the systemic infections, particularly typhoid and septicemia, in which the organisms are in the circulating blood, the bacteria themselves may be the pathogenic factors.

The kidneys are enlarged, in the severe cases sometimes to twice the natural size. They are soft and edematous. The tense capsule is not adherent. The smooth surface of the kidney is dark reddish-brown, or it may be pale or mottled and streaked with dilated vessels (Fig. 162).

FIG. 162



Acute parenchymatous nephritis: A, tubules showing cloudy swelling; B, congested Malpighian tuft; C, tubules with desquamated epithelium; D, Bowman's capsule infiltrated with leukocytes.

On section, the kidney shows a swollen and edematous cortex, corresponding in color to the surface. The normal striations are obscure. The whole pyramid or its boundary only is dark and congested.

The inflammatory lesions involve all the structures of the kidney. When the process is most intense in the tubules it is designated *tubular nephritis*; in the glomeruli, *glomerular* or *glomerulonephritis*; in the interstitial tissue, *productive* or *interstitial nephritis*.

Delafield recognizes an *acute exudative* and an *acute productive nephritis*.

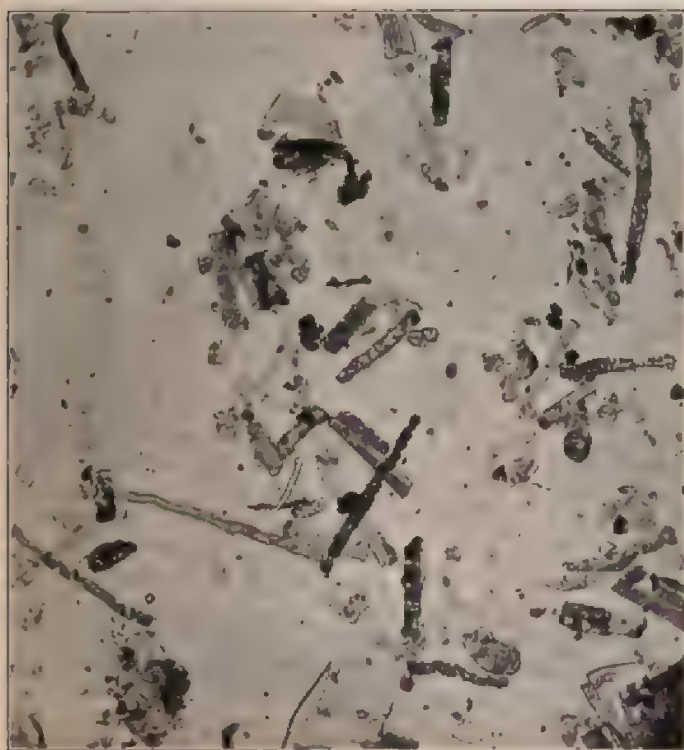
**Symptomatology.**—The onset of primary nephritis is often abrupt, the disease beginning with fever, headache, vomiting, restlessness, muscular twitchings, and, rarely, convulsions. While not usually an early symptom, dropsy occasionally first directs the attention of the



sician to the kidneys. In many cases the beginning is insidious, without marked renal symptoms, and will be detected only by the physician who always examines the urine of sick children. The temperature is irregular in type and not, as a rule, high. Fever may be absent during the first few days. In infants, however, a high temperature is often seen. Diarrhea has been noted in quite a number of cases.

In addition to the nervous symptoms above noted, severe cases with anemia show dulness and apathy, sometimes approaching coma. Holt states that anemia was a prominent symptom in his cases, and in several instances suggested the diagnosis.

FIG. 163



Urinary sediment in acute nephritis; hyaline, granular, and epithelial casts.

The urine may not be greatly decreased in amount, particularly in the beginning of the disease. Later it is often scant, and not infrequently oppressed. This symptom varies greatly in different cases and at different times in the course of an individual case. It is high colored and turbid, often red or smoky from the presence of blood. The specific gravity is high with scanty and low with abundant urine. Albumin is always present and in varying amounts, even enough to coagulate solid

on boiling. The amount may vary greatly from day to day, regardless of other symptoms.

Casts—hyaline, granular, epithelial—are always present, usually together with epithelial cells, debris, leukocytes, and blood cells (Fig. 163). In some cases there may be only an occasional granular or hyaline cast, and in others many of all varieties. Dropsy may be slight or marked. In one of my cases, an infant whose history follows, dropsy was very severe during the whole course of the disease.

The patient was a girl, aged twenty-three months. Prior to the attack she was a well child; no history of cold or any infection. On May 1st on awaking from her afternoon sleep her nurse noticed a puffiness of the face. Otherwise she was apparently well. She passed a restless night. The next day she was fretful, with no appetite, and in the evening she vomited. Another fretful night. She complained much of thirst, and vomited again on the morning of the third day. The edema was confined to the face below the eyes.

The child was seen by the attending physician on the evening of May 3d. The puffy face and vomiting led to an immediate examination of the urine. The nurse thought it somewhat diminished from the normal. A small specimen was obtained and found to contain 44 per cent. of albumin by bulk after precipitation, and great numbers of hyaline casts. The child's temperature was 99.2° F.; pulse 120. On the following morning the child was much better, and for five days she did not appear very ill. She was bright and playful. A slight edema then appeared in the feet and legs, which steadily increased. During this time the temperature did not rise above 99.2° F., and there was a fair amount of urine excreted. May 7th analysis showed the following: specific gravity, 1.028; reaction, acid; color, normal; albumin, 14 per cent. by bulk. Sediment: numerous hyaline and a few granular casts, one fatty cast; a few large and small round epithelia, and a few pus cells and red blood cells.

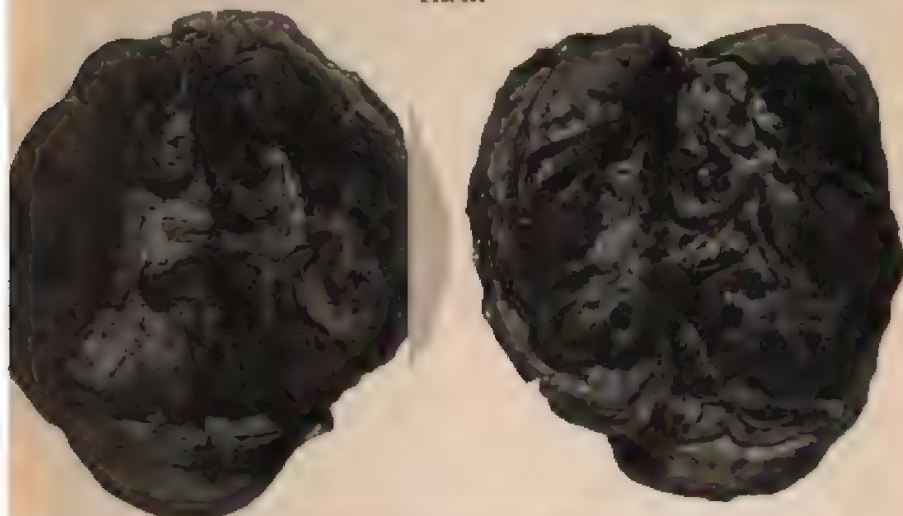
May 10th a twenty-four-hour specimen showed the following: quantity, 100 c.c.; specific gravity, 1.044; total solids, 10.25 gm.; color, normal, cloudy; albumin, 0.085 per cent. by weight; urea, 4.4 per cent. Microscope: many hyaline and a few granular casts; many pus cells and small, round epithelia, some showing fatty degeneration.

From May 10th to May 16th there was a steady increase in the edema. Except a lower specific gravity, the urine did not materially change in character. May 18th, symptoms were all increased in severity. Temperature, 102° to 103° F.; pulse, 150 to 160. Great general anasarca and ascites. Although the urine excretion was maintained, the child steadily grew worse and died May 20th, rather suddenly, of cardiac failure.

The following is a case in point: Child was born October 14th. Apparently healthy female, weighing seven pounds two ounces. Urine passed shortly after birth. Suppression on third day, with evening temperature of 103° F. Specimen of urine obtained on fifth day contained albumin in small quantity, uric acid crystals, blood cells,

and hyaline and granular casts. This was the only analysis made. During the course of the disease urine was passed only occasionally and in very small quantity. Fever of a remittent type continued, varying from 99° to 104° F. The child was drowsy, cried feebly at times, and showed muscular twitching. No dropsy. She gradually sank, and died on the sixteenth day. Autopsy revealed an acute nephritis of the hemorrhagic type (Fig. 164).

FIG. 164



Acute hemorrhagic nephritis in the newly born.

The affection lasts from two to four weeks. In infants acute primary nephritis runs a grave course.

Death occurs from acute uremia or from some one of the complications to which these patients are subject. Edema of the lungs or glottis, effusion into the serous cavities, pericarditis, endocarditis, pleurisy, pneumonia, and meningitis are most frequently noted.

The symptoms of nephritis secondary to one of the acute infections do not differ materially from those of the primary form. When it occurs during the height of the febrile process, the general symptoms are concealed by those of the primary disease, and with neglect of the examination of the urine it may escape recognition until suppression or a uremic accident rudely reminds the physician of his carelessness. Coming on when convalescence has begun, it is more readily recognized. There is a check in the progress of recovery. Fever returns, and with it vomiting, headache, prostration, and a scant, smoky urine. The temperature ranges from 100° to 102° F., rarely in severe cases reaching 104° or 105° F. Dropsy is usually present. Effusion into the serous cavities is not infrequent. Anemia is marked.

The urine is diminished in quantity, often suppressed. The specific gravity is low and the urea diminished. The color is dark red or smoky



from the presence of erythrocytes or hemoglobin. Albumin is always present, usually in large amount. The sediment contains casts in great numbers; epithelial, granular, hyaline, and blood casts are found during the early stage of the disease. Later the epithelial and blood casts disappear. Renal epithelium, red blood cells, and leukocytes are frequent.

The duration of a secondary nephritis is from two to four weeks. Approaching convalescence is marked by a decline in the constitutional symptoms and an increase in the excretion of urine, with a diminution in the albumin and the number and variety of the casts. Traces of albumin and a few hyaline casts may persist in the urine for a number of weeks. In children, left ventricle hypertrophy and accentuation of the aortic second sound develop early.

**Diagnosis.**—The diagnosis of acute nephritis rests upon the findings of urinary analysis. Chemical and microscopic examination of the urine should be made a routine measure in the diseases of children, as it is in the diseases of adult life. With proper examination of the urine, acute nephritis can be confused only with acute degeneration of the kidneys.

The distinguishing features of the two conditions are:

ACUTE NEPHRITIS.	ACUTE DEGENERATION.
May be primary or secondary.	Always secondary to an acute infection or intoxication.
Urine diminished, scant, or suppressed.	Urine diminished but slightly, or not at all.
Specific gravity normal, usually decreased.	Specific gravity normal or high.
Urea markedly decreased.	Urea normal.
Albumin always present in considerable amount.	Albumin present in small amount, usually only a trace.
Casts of all varieties, blood, pus, abundant epithelial cells, and epithelial debris.	Only a few hyaline casts present; granular rare; no blood or pus; only occasional epithelium or epithelial debris.

**Prognosis.**—Primary acute nephritis is a serious disease. Infants and young children frequently die. Older children less frequently succumb. Of the twenty-four cases in infants of Holt, sixteen died. Of the four cases seen by me during the last two years, all died. It should be remembered, however, that on account of the common neglect of urinary examination in infants many mild cases may run their course unrecognized. The inflammation is usually of the exudative type and recovery, when it occurs, is complete.

The immediate danger to life in secondary acute nephritis is not so great as in the primary form, death rarely resulting from the renal disease in the acute stage. As Delafield and others have shown, secondary nephritis, particularly when complicating scarlatina, is of the productive type, and the beginning of the chronic form of the disease. A guarded prognosis should be given in scarlatinal nephritis, even when apparent recovery has taken place, and the child should be kept under medical observation for several months or years.

Suppression of the urine, severe nervous symptoms, persistent vomiting, a severe anasarca, effusion into the serous cavities are unfavorable phenomena. The amount of albumin in the urine has no prognostic



significance. The amount of urine and the character of the sediment are better guides.

**Treatment.**—Much may be done during the course of the acute infectious diseases to prevent the onset of complicating nephritis. A simple fluid diet consisting largely of milk, with the avoidance of meat, should be kept up well into the convalescent stage. Bearing in mind the etiological relation of the micro-organisms and toxins of acute infections to nephritis, measures directed to the maintenance of abundant elimination are to be continued throughout the course of these diseases. Constipation should be carefully combated. Free elimination by the kidneys should be ensured by an abundance of fluid, often best administered in the form of alkaline carbonated waters, lemonade or other mild, agreeable beverages. The daily bath, warm or cool, is a grateful measure for the stimulation of metabolism and elimination. Every effort should be made to limit secondary streptococcus and other infections. The throat and nasopharynx are the chief portals of entry of these secondary infections; hence the importance of throat and nasopharyngeal cleanliness. The daily irrigation of the nose and throat with a mild alkaline and antiseptic solution is advisable in every case of acute infectious disease, however mild; in scarlatina and diphtheria it is essential.

With the onset of an acute nephritis the child should be given absolute rest in bed; even in the mildest cases this should be done and, except when convalescence is protracted, the patient should be kept at rest until the albuminuria ceases. When permitted to be up, the patient should be carefully guarded against excess, and the effect of exercise upon renal excretion noted by repeated urinary analyses. From the onset of the disease the total quantity of urine eliminated in each twenty-four hours and its specific gravity should be noted and recorded.

A daily warm bath stimulates diaphoresis and diuresis, and is to be recommended.

A strict regulation of the diet with the careful adjustment of the proteid content of the food to the demands of nutrition and to the functional activity of the diseased kidneys and the avoidance of substances difficult to excrete is essential.

von Noorden and Chittenden have shown that a man weighing 154 pounds (70 kilos) will maintain his nitrogenous equilibrium under conditions of moderate activity with a diet containing from 50 to 60 grams of proteid. From this it may be estimated that a child aged four years may maintain a fair nutrition during the short period of an acute illness with food containing 25 grams of proteid; a child of eight years with 30 grams; and a child of twelve years with 35 grams. The total caloric need of these children under such circumstances will not exceed 1000 calories at four years; 1200 calories at eight years; and 1400 calories at twelve years.

The kidney excretes with difficulty urea, creatinin, phosphates and water. Proteid food yields urea; creatinin is a large constituent of

meat extracts and broths; phosphoric acid is contained in large amounts in meats, yolks of eggs, milk and many vegetables.

With these data it is not difficult to construct a dietary suitable to an acute nephritis.

Milk has long been considered the ideal food in nephritis. It should not be the sole food, but may be the chief proteid-containing article of diet. Each 100 c.c. of milk contain 4 grams of proteid. 600 c.c. (20 oz.) will supply the proteid need of a child of four years; 750 c.c. (25 oz.) of a child of eight years; and 900 c.c. (30 oz.) of a child of twelve years. The caloric yield, however, of these quantities of milk is not sufficient. Each 100 c.c. of milk supply approximately but 70 calories, making the total yield of the quantity of milk which contains the necessary proteid, about one-half of that necessary for nutrition.

The deficiency of calories may be made up by the use of foods rich in carbohydrates and fat and containing a small per cent. of proteid, such as cream, butter, sugar and the cereal products. These foods may supplement the milk in the dietary. For example, 16 oz. milk, 4 oz. cream, 1 oz. butter, 2 oz. of bread and 1 oz. of sugar contain approximately the 25 grams of proteid, and will yield approximately the 1000 calories necessary for the daily nutrition of a child aged four years. During the early stage of the disease milk, diluted cream and the cereals gruels should constitute the diet—later the solids may be added. Return to a usual meat containing diet should not be made until convalescence is well along. Additional articles of food should be given carefully and the effect on renal excretion noted. An increase or a return of the albuminuria will caution still further delay.

A brisk calomel purge should begin the treatment of nephritis, and subsequently throughout the disease a thorough evacuation of the bowels should be ensured daily by the administration, if necessary, of an efficient laxative. Children usually take, without trouble, citrate of magnesia, compound licorice or jalap powder, and sometimes the sulphated mineral waters.

With this hygienic and dietetic treatment guarding the inflamed kidney from irritation and excessive work, acute nephritis usually terminates in recovery.

In severe cases suppression of the urine with its sequels, dropsy and uremia, will demand active treatment. In the early stage the kidneys are engorged with blood and their function is arrested. They are practically impervious to water.

To relieve engorgement and aid in the restoration of secretion, counterirritation may be made over the kidney with dry cups, and this followed by the application of a hot compress or poultice.

When engorged the kidneys eliminate water with difficulty, consequently the fluid should be restricted to the amount absolutely necessary to maintain nutrition and should not exceed from 1000 c.c. to 1200 c.c., depending upon the age of the child. Most of this will be given in the milk and cream of the food.

The bowels and skin should be called upon to relieve the kidneys of the work of elimination. Free catharsis is always indicated; diaphoresis chiefly when dropsy is present. The skin can only eliminate water; the excretory solids must be eliminated by the bowels. The hot pack is the most reliable diaphoretic. In an emergency pilocarpin may be given.

Rectal irrigation with normal saline solution at a temperature of 106° to 108° F., with a double tube, is efficient in relieving engorgement and restoring secretion.

For uremic symptoms with high pulse tension, nitroglycerin 0.00021 gm. to 0.00032 gm. ( $\frac{1}{3000}$  to  $\frac{1}{2000}$  gr.) every hour for four or five doses is useful. Chloral, by rectum 0.065 gm. to 0.13 gm. (1 to 2 gr.) for each year and repeated in from two to three hours, should be given when nervous symptoms are present. For convulsions, chloral, as above, or morphine hypodermically may be given. Chloroform carefully administered hastens relaxation and gives the more slowly acting sedatives time to produce their effects. In grave cases the abstraction of from 90 c.c. to 150 c.c. (3 to 5 oz.) of blood, with the introduction of normal saline solution by hypodermoclysis or enteroclysis, may save life.

The onset of anemia is rapid, and early in convalescence demands iron. Recovery in severe cases is slow and requires the most careful guidance. Continuation of the dieting and the hygienic precautions above laid out may be necessary for several weeks or more. Whenever possible, the winter in a dry, warm climate is advisable for two or three years. The possibility of the insidious development of a chronic nephritis should never be forgotten.

### CHRONIC NEPHRITIS.

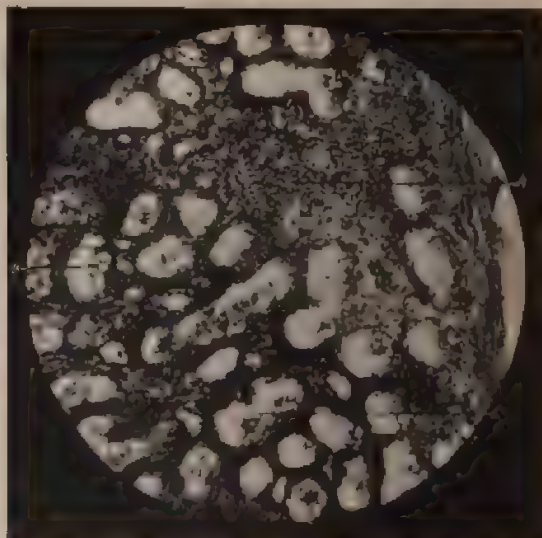
Two forms of chronic nephritis have received general recognition: 1. Chronic Parenchymatous or Diffuse Nephritis. 2. Chronic Interstitial Nephritis.

In both of these forms of nephritis there are changes in the epithelium, the glomeruli, and the stroma. The predominance of the changes in one or the other of these elements of the kidney does not influence the clinical symptoms. Delafield holds that the essential difference in the pathological processes, the difference that determines the clinical course of the disease, is the presence or the absence of exudation. "In all these kidneys two changes are constant—productive inflammation of the glomeruli and stroma and desquamation of the renal epithelium. The only real difference between the kidneys is whether, besides the growth of new tissue and desquamation of the renal epithelium, there is or is not an exudation of serum from the bloodvessels of the kidneys." So that Delafield terms the two pathological varieties of chronic nephritis "*chronic productive nephritis with exudation*" and "*chronic productive nephritis without exudation.*"



**Chronic Parenchymatous Nephritis. Etiology.**—This form of chronic nephritis is the one more frequently observed in childhood. Compared with adult life, it is rare in childhood. It is most frequently seen at the age of five years, while in early childhood and in infancy it is very rare. As a rule, it occurs in children as a sequel to acute nephritis of the productive type, occurring as a complication of scarlatina. The history of these cases often shows a sequence of several of the acute infections occurring during the two or three years preceding the onset of the nephritis. Syphilis, tuberculosis, chronic endocarditis or chronic suppurations are occasional etiological antecedents. Rarely a case is met that is chronic from the onset and can be traced to no adequate cause.

FIG. 165



Chronic diffuse parenchymatous nephritis: A, young connective tissue; B, dilated tubule with flattened epithelium; C, tubule with partially desquamated epithelium.

**Pathology.**—The gross and microscopic pathological anatomy of the kidney in the chronic nephritis of childhood is the same as seen in the adult. The large white kidney is most frequently seen and is sometimes enormously enlarged. Ashby and Wright cite a case of a girl of twelve years, in whom the two kidneys together weighed two and three-quarter ounces, and the left kidney measured six inches in length. In cases in which the fatal termination is long delayed the small white kidney sometimes is seen (Fig. 165).

**Symptomatology.**—An acute nephritis may pass on to the chronic form without an intermission in the symptoms, or, after an intermission during which the patient appears in good health, persistent symptoms develop. In a certain number of cases two or more attacks



of what appears to be acute nephritis precede the fixation of the chronic disease. In such cases it is probable that a mild productive inflammation without exudation is continuous, and that the so-called acute attacks are exacerbations in the symptoms that mark extensions or renewals of the exudative process. Occasionally no history of an acute nephritis can be obtained.

Dropsy is a characteristic symptom of chronic parenchymatous nephritis. It may appear as a localized or a general edema or as an effusion into one of the serous cavities. Anemia is another marked symptom and with the dropsy give the characteristic puffy, pasty skin of chronic Bright's disease.

Digestive disturbances are prominent—anorexia, indigestion, and attacks of vomiting or diarrhea. Headache, insomnia, dyspnea, and other uremic phenomena appear from time to time. Persistent debility with anemia are sometimes the only symptoms to direct the attention to the kidneys. Cardiac hypertrophy is present in all cases that have continued for any length of time. In children, retinal changes are not common.

The urine is often normal in amount, often diminished, sometimes increased. The specific gravity is usually low and the urea excretion diminished. Albumin is always present, usually in moderate or large amount, often 0.5 per cent. or more by weight. During the intervals between exacerbations the quantity, specific gravity, and urinary solids may be but slightly or not at all below the normal; at these times only a trace of albumin may be present.

The urine is often cloudy and contains an abundant sediment. The microscope shows many epithelial cells and much epithelial debris with hyaline, granular, and epithelial casts. Fatty casts, fatty renal cells, and fat globules are often abundant. Blood and pus may be found, the blood particularly during the exacerbations. At these times analysis shows a urine similar to that found in acute nephritis.

The course of the disease is very irregular and it is marked by repeated remissions and exacerbations. It usually covers a period of several years. Some patients have the disease during all of childhood and adolescence and succumb in early adult life. Exhaustion, acute uremia, pneumonia, and other complicating inflammations are the immediate causes of death.

**Diagnosis.**—With the realization of the necessity for the examination of the urine of every sick child, chronic nephritis will not escape detection. Chronic digestive disturbances, a persistent debility, anemia, and dropsy should always direct attention to the kidneys. The presence of albumin and casts with defective elimination will reveal the nature of the trouble.

**Prognosis.**—The prognosis of chronic parenchymatous nephritis, while not so grave in children as in adults, is decidedly unfavorable. After continuing for several months some cases apparently recover. In such cases the pathological process is arrested, leaving healthy kidney tissue sufficient to carry on excretion. Other cases after several years of

quiescence relapse, and die from renal insufficiency. The majority of the cases progress with remissions and exacerbations to a fatal termination. An abundant urine of persistently low specific gravity is indicative of a large connective-tissue growth in the cortex, or waxy degeneration of the glomerular vessels and is of unfavorable significance.

**Treatment.**—Taken early, before extensive productive and degenerative changes have taken place in the kidney, much may be done by medical treatment to aid the arrest of the disease. The same principles of treatment are applicable to children as to adults. Hygienic measures should occupy the first place. Repeated exposures, digestive disturbances and disorders of metabolism, with their accompanying engorgements of the kidney, should be carefully avoided. Woollen undergarments will protect from surface chilling, as exposure to cold and wet is to be avoided. If it is possible, an early removal of the patient to a dry, warm climate should be advised. Removal to a Northern climate should not be attempted for two or three years. An out-of-door life with exercise, but never to exhaustion, is important.

During exacerbations and whenever there are evidences of renal insufficiency rest in bed should be enjoined. The diet should be liberal, but simple and easily digested, and carefully adjusted to the total daily amount of proteids, carbohydrates, and fats to the needs of nutrition and the capacity of digestion. Vegetable and milk proteids are the least irritating to the kidneys, and a milk and cereal diet is an ideal one in chronic nephritis. The milk may be given diluted with an alkaline mineral water like Vichy, or with the cereal gruels. Light, succulent vegetables and cooked fruit may be given in moderation. Raw fruit is better avoided; it is slow of digestion, contains but little nutriment, and favors the production of acid indigestion. Eggs, fish, and fowl may be given sparingly. Red meats should be given with great caution or not at all. When it is difficult to give sufficient proteid in the milk and vegetables to repair tissue waste, scraped meat, beef-juice, and light broths may be used with advantage. The stock soups, beef-tea, and beef-extract are to be avoided. An amount of fluid sufficient to ensure free elimination should be insisted upon.

The condition of the stomach and bowels should be carefully watched. A daily free movement of the bowels is essential, as a day or two of intestinal intoxication may precipitate an attack of uremia or an exacerbation of the exudative process. Irrigation of the colon at these times is of great value. Cutaneous elimination should be favored by a daily warm bath.

The debility and anemia demand the more or less prolonged use of tonics. Iron in small doses, and quinine and strychnine are of value. Large doses of iron do harm by deranging the digestion.

**Surgical Treatment.**—Dr. George M. Edebohl, of New York, proposed in 1899 to treat chronic nephritis by renal decapsulation, and the reports of Dr. Edebohl and others show what seem to be remarkable results from this operation.

In May, 1902, Dr. A. Caillé, of New York, reported in full to the American Pediatric Society a successful case in a girl aged five years. The child had been under medical observation for three years, and at the time of the operation, February 15, 1902, presented the clinical characteristics and urinary findings of advanced parenchymatous nephritis. The case was carefully studied by Dr. Caillé, both before and after the operation. At the time of his first report, three months after the operation, the child was steadily improving, although she was still anemic, and the urine showed albumin and casts. At the meeting of the American Pediatric Society in June, 1904, Dr. Caillé reported the recovery complete. Dr. T. M. Rotch reported to the same Society a case of advanced nephritis with operation. The child, a boy aged ten years, showed a temporary improvement, but died nineteen days after the operation, with symptoms of pulmonary edema and cardiac exhaustion. Dr. Tyson (*Practice of Medicine*) reports one case in a girl aged ten years. The child had a very severe chronic diffuse nephritis that had lasted over four years. At the time of operation she was very weak and had general anasarca and ascites. At the first operation one kidney was decorticated. Dr. Tyson says that the result of this "may be truly called marvellous." A month after the operation the child was apparently well, although albumin and casts were still abundant in the urine. Following operation on the second kidney the child made a prompt recovery, and at the time of the report she was apparently well.

Such results are remarkable and make surgical interference in chronic parenchymatous nephritis that has resisted medicinal treatment more than justifiable. In commenting on his case, Dr. Caillé states: "From this and other cases which have come under my observation, I should be willing to advise inspection of the kidneys through lumbar incision in cases in which an acute nephritis, not secondary to heart lesions, does not clear up in a reasonable time, say six months, and would, furthermore, advise decapsulation of one or both kidneys should they appear swollen and enlarged, with the hope of preventing the acute nephritis from becoming chronic."

**Chronic Interstitial Nephritis.**—Chronic interstitial nephritis is a very rare disease in early life. Only a few cases are found reported in the literature of diseases of children. Gull and Sutton recorded the first case in 1872. Ashby and Wright met with two cases which came under observation only a few days before death. Dickenson was able to collect five cases occurring under the age of twelve years. Other cases are mentioned by Barlow, Goodhardt, and Bartels. Tyson has never met with a case in a child. Guthrie (*London Lancet*, 1897) reported seven cases in which the diagnosis was confirmed by autopsy. Sawyer, in a recent article, reports a study of twenty-four cases.

The recognition of the possibility of contracted kidney in early life, and the more frequent investigation of the urine of children suffering from obscure chronic disease may bring to light a greater frequency of chronic interstitial nephritis than is now suspected (Fig. 166).





symptoms to fix the attention. Guthrie found these patients undersized and wasted. The wasting is of long standing and usually is attributed to other causes. With the wasting is a dry, coarse, and inelastic skin.

Eustace Smith states that the gums and conjunctive are markedly pale, while a dusky flush of the face from general capillary congestion masks the anemia. The pigmentation varies from a mere sallowness to a marked bronzing of the skin, distributed generally or in patches. Dropsy is usually absent. Exceptionally it is present for a short time before death. During periods of intercurrent renal congestion or exudative inflammation, marked by scanty, albuminous and bloody urine, it may be transiently present. Chronic indigestion, with occasional attacks of vomiting and diarrhea, or constipation and abdominal pain are common. Excessive thirst is frequent. Headache, vertigo, dyspnea, and convulsions are the most common nervous symptoms. Visual disturbances, such as amaurosis or diplopia, and cerebral hemorrhage, have been noted. These children are sensitive to exposure, and bronchitis and bronchopneumonia are frequent complications. Edema of the lungs and asthma may occur. Cardiovascular hypertrophy with high arterial tension is usually present. With advanced cardiac and vascular changes precordial pain and distress are common.

The urine in interstitial nephritis is increased in quantity, pale, and of low specific gravity. Sawyer has noted that in some instances there was a history of polyuria from birth. Albumin is usually present, but in small quantity, often only a trace. As in the adult, it may be absent for long periods, or it may come and go. Acute exacerbations are always marked by an increased albuminuria.

The sediment is light and contains hyaline casts in small numbers. Careful search of the centrifugalized urine is often necessary to demonstrate them. Occasionally granular casts are found, and during periods of increased renal engorgement blood, pus cells, and epithelial debris may be present.

**Diagnosis.**—From the above it is plain that chronic interstitial nephritis should be suspected in every case of chronic intestinal derangement and grave and persistent interference with nutrition in childhood. A polyuria should always be carefully investigated. The presence of the above-noted symptoms, together with a polyuria, renal insufficiency, persistent, mild, continuous or intermittent albuminuria, and the presence of hyaline or granular casts would determine the diagnosis. In children, although not so frequently as in adults, occasional hyaline casts are found in conditions of renal irritation, such as are seen in lithemia and other disturbances of metabolism. In these conditions there is the absence of polyuria, and the urine is of normal or high specific gravity and color. Only by repeated urinary analyses and prolonged clinical study can a certain diagnosis be made.

**Prognosis.** The course of chronic interstitial nephritis is long and its termination uniformly unfavorable. Sawyer is of the opinion that the disease may begin in childhood, subsequently undergo arrest, and in

adult life, under favorable conditions, start up afresh and continue the well-known progressive course.

**Treatment.** The treatment of the disease is principally dietetic and hygienic. The diet should be milk, but not a great amount at a time, as the digestive strength may be impaired and the cardiovascular symptoms increased by an overabundant liquid diet. Water is needed by the system, and it should be taken between meals. Cereals, cooked from oatmeal, and green vegetables may be added to the diet if the digestion is not impaired. These children require fat and should be given butter and fat bacon. The skin must be kept protected by woollen undergarments. It is best to bathe the children with tepid water rather than to attempt sponging with cold water. Sunshine and a warm climate will add to the comfort and strength of these cases, while cold winds and dampness will render them liable to intercurrent affections.

Medicines are not of much service. The action of the liver should be watched and an occasional dose of a mercurial may be administered. With high arterial tension nitroglycerin is helpful, and small doses of iodide of potash are of benefit. Both of these drugs may be continued for some time.

### PERINEPHRITIS.

Perinephritis is an inflammation of the loose connective and adipose tissues surrounding the kidney. While not of frequent occurrence, it should always be kept in mind when dealing with obscure diseases of the abdomen.

**Etiology.**—The disease may be either primary or secondary. The primary form, more common in children, may be due to traumatism or exposure. The etiology of many cases is obscure. The secondary form of the disease may result from extension of any neighboring infectious process, especially in the kidney. It may occur as a complication or sequel of any of the acute infectious diseases.

**Pathology.**—The infection, when not tuberculous, is usually by the ordinary pus organisms. The disease may terminate in resolution or in suppuration. When pus forms, the abscess cavity may be small or very large. As much as two or three pints of pus have been evacuated from one such abscess. There is always a tendency for the pus to burrow, and it is only the smallest abscesses that are well walled off. If left unopened, the abscess may break through into any part of the intestinal tract or into the peritoneal cavity. It may perforate the diaphragm and pus be coughed up. It may come to the surface in the groin, the lumbar region, or the iliocostal space. Usually only one side is affected.

**Symptoms.**—In children the onset is commonly abrupt or it may be gradual. There are fever and chills, and pain which may be referred to the loin, to the groin, or down the leg. Tenderness in the region of the affected kidney is generally present early. As the inflammation spreads there will be lameness of the leg. The thigh is commonly drawn up

and extension is painful. There may be deviation of the spine with the concavity toward the affected side. Later, a tumor can be made out in the loin, and there may be infiltration of the skin in the ilio-costal space. The constitutional symptoms later may become severe. When the onset is gradual, the pain, tenderness, and stiffness may precede by several days the appearance of constitutional symptoms. No urinary symptoms are to be expected from a primary perinephritis.

**Diagnosis.**—Hip-joint disease may be excluded by a careful examination. In perirenal abscess there is no general joint tenderness and no pain connected with any motion of the thigh except that of extension. An abscess may be fully formed within two or three weeks after the first symptoms if there is a tumor. The opposite prevails in hip-joint disease; the onset is insidious instead of acute. Often an entire year elapses before the development of the abscess; all motions of the hip-joint are painful. Deformity in hip-joint disease increases much more slowly.

One must always exclude the angular deformity and spinal symptoms of Pott's disease. When the pus burrows through the diaphragm and appears in the sputum, a diagnosis of empyema may be made. Baginsky reports a case in which a perinephritis was secondary to a purulent pleuritis, probably tuberculous.

The exploring needle and the high leukocyte count will aid in the detection of pus when fluctuation cannot be obtained.

**Prognosis.**—In primary cases the prognosis is good when the condition is recognized early. Of 36 cases observed by Gibney, referred to by Holt, all recovered. The process may terminate by resolution, in which case soreness and stiffness in the back disappear very slowly.

In cases secondary to severe local processes the prognosis is not so good. When there is spontaneous opening to the exterior, healing is obstinate.

**Treatment.**—Rest in bed, hot fomentations, or the ice-bag to the affected area are primary indications. Abscesses should be watched for and promptly opened with due surgical precautions. Otherwise, the treatment is symptomatic.

### LITHIASIS.

The formation of concretions in the urinary tract may be due to changes in the composition of the urine or to interference with its excretion. The increased metabolism of infancy and childhood predisposes to this disease. Most cases in childhood occur between the ages of two and ten, but large concretions have been found in the bladder at birth, and cases may be met at any period later.

Calculi occur much more frequently in some localities than in others. In China and Asia Minor the condition is exceedingly common, and the amount of calcareous salts in the drinking water is, undoubtedly, an etiological factor. A family history of gout or rheumatism is very common.



**Uric Acid Infarcts.**—In 40 per cent. of autopsies on infants less than two weeks old, there is found a condition, first described by Virchow, which he named *uric acid infarct*. The cause is the formation of uric acid in the kidney before there is sufficient water ingested to carry it off. The infarct appears in the gross specimen as fine, reddish-brown lines, radiating from the pelvis of the kidney. The microscope shows the characteristic uric acid crystals. There is often a small deposit of these crystals in the pelvis of the kidney. When diuresis is established this deposit is washed out and appears upon the diaper as a reddish deposit. There may be slight irritation of the kidney, and temporary anuria is common, generally easily relieved by hot applications and the ingestion of plenty of water. Rarely a severe renal congestion with suppression is observed.

**Renal Calculus.**—The uric acid deposits of the newborn may form a nucleus for the formation of renal calculus. The chemistry of renal calculus and the mechanism of formation in the child is the same as in the adult. In the kidney cortex itself a stone commonly gives no symptoms, unless large, when pain and tenderness in the kidney region may lead one to suspect its presence. A history of the passage of small calculi is significant, and the skiagraph now offers a method of positive diagnosis. In the pelvis of the kidney irritation of the stone may lead to a pyelitis, and by impaction in the upper opening of the ureter cause pyonephrosis or hydronephrosis.

**Renal Colic.**—When a calculus is of such a size that it passes down the ureter only with difficulty it may produce the most excruciating pain. During an attack the child screams with pain, which is periodic in character, resembling severe intestinal colic. The face is anxious, flushed, and covered with perspiration. The child makes frantic efforts to urinate and succeeds in passing only a few drops, often containing blood and mucus. Convulsions may occur. In male children the testicle of the affected side will be drawn up. Pain commonly ceases when the stone reaches the bladder. Older children will describe the pain as radiating backward and down the thigh from the affected kidney. In infants immediate diagnosis from intestinal colic is often impossible; the retracted testicle may be suggestive. Urinary examination is necessary to a positive diagnosis. If the urine be examined immediately after such an attack, it will be found to contain blood, a considerable amount of epithelium, and often pus cells. It may be either alkaline or acid; in the latter case it usually contains uric acid crystals. In severe kidney irritation there are hyaline casts.

R. P., aged 11 years, a sturdy boy. For a few weeks he had a disturbed digestion with abdominal intestinal fermentation. While in school, about nine o'clock in the morning, he was suddenly taken with a severe pain in the left loin, the pain radiating down the side round across to the median line of the abdomen. When seen two hours later he was still in great pain. Pulse and temperature normal. He had vomited. The back over the left kidney was very tender. No tenderness over the front of the abdomen. No retraction of the testicle. Pain was



not reflected down the thigh. The urine contained a trace of albumin and the centrifugal sediment showed numerous red blood cells and many large calcium oxalate crystals. A hot rectal irrigation and hot stupes eased the pain, and by evening it had entirely disappeared, leaving a tenderness that faded by the following day. An abundant flow of urine followed several hours of scant excretion.

**Vesical Calculus.**—The symptoms of stone in the bladder are somewhat different in small children from those in the adult, chiefly owing to the shape of the bladder. In the narrow, pyramidal bladder of the child, the stone being in the most dependent part, it constantly assaults the sensitive vesical neck, producing many reflex symptoms; while in adults it may lie farther back, causing much less irritation and no interference with the flow of the urine. There may be, in the child, painful urination, interruption of the stream, retention, incontinence, hematuria, cystitis, albuminuria, reflex pains, rectal tenesmus, and prolapse. The pain may be reflected to the end of the penis, and a disposition to pull the prepuce is often noted.

**Diagnosis.**—In vesical calculus this is made positive by the sound. An anesthetic is generally necessary. In passing the sound, one must remember that the angle in the urethra behind the triangular ligament is much more acute in children than in adults.

**Prognosis.**—Under good conditions the results of operation for renal calculi have been encouraging. Before operation is attempted, it should be positively ascertained, if possible, that the other kidney is not diseased. This may be determined by catheterization of the ureter. Calculous pyelitis, if unoperated, may lead to perforation, generally behind, with the formation of a fistula in the lumbar region. Perforation into the peritoneal cavity with fatal result has been reported. The results of calculi impacted in the ureter may be serious unless treatment is prompt.

**Treatment.**—No one now expects to dissolve a stone by medicinal treatment. When once formed, whether in kidney, ureter, bladder, or urethra, curative treatment must be surgical.

Gravel may be washed out. The administration of large quantities of fluid is the most essential part of the medicinal treatment. The alkaline mineral waters are commonly prescribed; of these, Vichy is one of the best. Careful examination of the urine is necessary, however, to intelligent treatment. When an alkaline urine is depositing a phosphatic layer around a stone, alkalies are contraindicated. In this case it must also be borne in mind that urotropin, so often prescribed, is active only in acid urine. For the irritation from stone in the kidney, glycerin has been recommended—4 c.c. to 12 c.c. (1 to 3 dr.)—in solution and may be given every four hours. For renal colic relief of pain is imperative and demands opium.

Where the tendency to lithiasis is shown, without further evidence of disease than a heavy, acid urine, a diet with a minimum amount of meat, and plenty of milk, should be ordered. With a distinct gouty or rheumatic history, treatment should be by alkalies and salicylate of soda.

## TUMORS OF THE KIDNEY.

**Benign Tumors.**—The literature shows that benign tumors of the kidney in children are very rare. Aldibert, out of fifty-one collated cases, found but three benign growths. As a rule, they grow to only very moderate size and give rise to few, if any, symptoms. A limited size, slow growth, and the absence of the constitutional symptoms that inevitably attend malignant growths would aid in a differential diagnosis.

**Malignant Tumors.**—Malignant tumors of the kidney in early life are of sufficient frequency to make them of great clinical importance. The recent studies of Birch-Hirschfeld, Walker, McWilliams and others have given us a clear conception of the pathological relations of these interesting growths.

They have been variously described as carcinomata, sarcomata, endotheliomata, rhabdomyosarcomata, etc. Birch-Hirschfeld demonstrates that they properly belong to a distinct class, which he designates embryonal adenosarcomata. He and other observers recognize carcinomata among the primary malignant tumors of children, although they are extremely rare. Walker thinks it doubtful if carcinoma ever occurs in young children.

Eberth was the first to demonstrate that the embryonal adenosarcoma takes its origin from remnants of the Wolffian body. These tumors always develop inside the kidney. The kidney tissue proper, however, does not take part in the process, but becomes compressed and atrophied as the tumor grows. The tumor develops from the pelvic region, often splitting the kidney at this point so that what remains of the kidney rests on the tumor like a flat cap (Strong).

The left kidney is more frequently affected than the right. Occasionally both kidneys are the seat of growths. At first the growth is slow; later it is extremely rapid. Metastasis is late and occurs in about one-half to one-third of the cases. The liver, the lungs, the other kidney, and the mesenteric nodes, and occasionally the colon, small intestines, and adrenals may be invaded. The infrequent involvement of the ureter and bladder is notable. The metastases are sarcomatous.

**Etiology.** *Age.*—These tumors have been found in the seventh and eighth months of fetal life. They are most frequent between the ages of six months and four years. About 80 per cent. occur under the age of four years, and 20 per cent. under the age of one year. Between the sixth and ninth years they are very rare, and above nine years are practically unknown.

The reported cases seem to be about equally divided between males and females. Birch-Hirschfeld considers them more frequent in females. Heredity appears to have no influence. Among immediate causes, infectious diseases, traumatism, chronic irritations, as from calculus, have been cited, but the etiological relationship is not clear.

**Symptomatology.**—The characteristic and most commonly observed symptoms of renal sarcoma are tumor, hematuria, pain, and cachexia.

**Tumor.**—In from one-half to one-third of the reported cases this is the initial symptom. Occasionally it is accidentally discovered. When small, the tumor is first detected in the lumbar region. Enlarging often with great rapidity, it extends downward and inward, the upper border reaching the median line just above the umbilicus and curving down to the iliac fossa. Enlargement may continue until the whole abdominal cavity is filled. Small and moderate sized tumors are movable on palpation and with respiration. Tenderness is uncommon. The tumor may be round, oval, kidney-shaped, or nodular. The surface is smooth. Small tumors are hard; the large ones often soft—almost fluctuating. The colon usually lies between the tumor and the abdominal wall, and can be demonstrated by percussion when distended, or palpation when flattened and empty. This is a very important diagnostic sign.

**Hematuria**, abundant or manifest only by the microscope, occurs in about one-third to one-fourth of the cases. It is often the initial symptom. Of 50 collected cases by Lebert, hematuria was the first symptom. It may occur once, or repeatedly, at longer or shorter intervals.

**Pain.**—Pain is often an early symptom. Usually it is a more or less continuous dull ache; often it is sharp, severe, or intermittent. Intense paroxysms have been noted. It may be confined to the side or lumbar region, or it may extend to the hip, thigh, or leg. Occasionally it shoots down to the testicle. Capsule tension from rapid growth, pressure on neighboring nerves, ureteral obstruction, peritonitis, and spinal erosion are factors in pain production. Simple discomfort from the size and weight of the tumor is often pronounced. A recent case had no pain.

**Cachexia.**—Constitutional symptoms are absent during the early part of the disease. Later, weakness, emaciation, anemia, loss of appetite, rapid pulse, and symptoms produced by pressure and interference with neighboring organs supervene. Emaciation, often rapid and extreme, results from interference with digestion, and from the absorption of the toxic products of tumor metabolism. Anemia is often marked. Few careful blood analyses have been made. Edsall has noted moderate leukocytosis.

**The Urine.**—A renal hematuria, as above cited, is the most characteristic urinary symptom. The urine is usually acid, with the specific gravity from 1.010 to 1.040. Sugar has not been found. Albumin occasionally is present. Urea is, as a rule, diminished. Rarely hyaline and granular casts and pus have been observed. In the pus cases a complicating cystitis was present. Blood clots, necrotic shreds, and tumor elements have been observed.

Various pressure symptoms attend the later period in the clinical history of the malady. Displacements of the stomach, liver, and other organs, with interference with their functions, are common. Vomiting, constipation, diarrhea that sometimes is bloody, jaundice from common-duct obstruction, pigmentation from adrenal invasion, all have been observed. Cough and dyspnea from pulmonary metastasis have been noted (Osler). Ascites and edema of the lower extremities are late



symptoms. Suddenly appearing varicocele and hydrocele are recorded. Uremia with convulsions, headache, vomiting, and coma have been observed. Death results from exhaustion or intercurrent complications.

**Diagnosis.**—Successful surgical treatment of sarcoma of the kidney depends, in large measure, upon an early diagnosis. The insidious development of this growth makes its early recognition exceedingly difficult. Persistent abdominal or lumbar pain, or a hematuria, demands a thorough physical exploration of the abdomen, under an anesthetic if necessary.

Differentiation has to be made between enlargements of the kidney other than sarcomata and enlargements and tumors of neighboring organs. Among the most frequent conditions to be differentiated from renal sarcoma are tumors and enlargements of the liver and spleen, malignant growths of the retroperitoneal lymph nodes, ovarian tumors, perirenal abscess, congenital cysts of the kidney, hydronephrosis, and pyonephrosis. Of the tumors of the abdomen in children, sarcoma is the most frequent. Osler says that large, solid, abdominal tumors in children are almost always sarcomata. Sarcomata grow with great rapidity, and during the early part of their development are not attended by constitutional symptoms. They grow from the lumbar region, downward and inward toward the iliac fossa. Tumors of the liver are very rare. Fatty and other enlargements of the liver are easily recognized from their location in the hypochondriac region, superficial position, and the sharp border.

Splenic enlargements, although not frequently met with, may closely simulate kidney sarcoma. The spleen does not enlarge downward into the iliac fossa and afterward toward the umbilicus. A sharp, notched border is characteristic. The colon lies *behind* a splenic enlargement and *in front* of a sarcoma of the kidney. Inflation of the bowel may be necessary to determine the relations of the colon to an abdominal tumor. Blood changes, often profound, are manifest in splenic tumors. Retroperitoneal tumors, when large, are difficult of differentiation. Their central position in the abdomen is characteristic. Ovarian tumors, very rare in children, grow from the pelvis upward.

In all these conditions hematuria and other urinary symptoms are absent.

Congenital cysts of the kidney are large, movable, and fluctuating. Constitutional symptoms are absent.

Hydronephrosis presents a movable, fluctuating tumor. Its disappearance after a large discharge of urine is characteristic. Primary tuberculosis of the kidney offers great difficulty in diagnosis. The constitutional symptoms and the urinary findings are suggestive; the demonstration of the tubercle bacillus is conclusive. Perirenal abscess and pyonephrosis, both very rare, are attended by pain and tenderness, swelling in the back, fever, and other constitutional symptoms. Aspiration may assist in the differentiation of cysts, hydronephrosis, pyonephrosis, and perirenal abscess. It is not, however, without dan-



In all cases in which a diagnosis cannot be made, and particularly if there be present suspicious urinary findings, an exploratory operation should be done.

**Prognosis.**—Without operation, malignant tumors of the kidney are invariably fatal. In 142 cases collected by Walker, the average duration of life without operation was about eight months. Death may occur inside of two months, and it has been delayed two and one-half years. The soft tumors grow more rapidly and kill more quickly than hard tumors.

**Treatment.**—The treatment is divided into medical and surgical. Medical treatment is palliative, as no remedy is at present known that controls, in the least degree, the progress of the disease. Coley's serum may be used; no successes have, however, been reported.

The prominent symptoms demanding treatment are pain and hematuria. For pain produced by nerve pressure, hot fomentations are of value. A large, thick, hot-water compress covered by an impervious dressing may be bound around the abdomen and changed every two to four hours. For the pain of local peritonitis and capsule tension the ice-bag is efficient. In the later stages and in the severe paroxysms due to ureter obstruction, anodynes are imperative. Codeine and morphine hypodermically; phenacetin and antipyrin by the stomach are most efficient. Hematuria only exceptionally demands treatment. The pain of capsule tension is not infrequently relieved by hemorrhage into the pelvis of the kidney. When the bleeding is excessive and persistent, ergot, 2 c.c. ( $\frac{1}{2}$  dr.) of the fluid extract every three hours, or solution of ferric alum, may be given. The ice-bag is often efficient. Adrenalin chloride, 0.60 to 1.25 c.c. (10 to 20 min.) of the 1:1000 solution, may be administered at frequent intervals hypodermically or by the stomach. Proper hygienic surroundings, good nursing, abundant easily digested food, and tonic medication prolong life. Pressure symptoms and intercurrent complications must be treated on general principles.

**Surgical Treatment.**—The opinions of surgeons as to the justifiability of operative interference for renal sarcoma is not uniform. Gross thought the operation unjustifiable. Aldibert, considering the high operative and ultimate mortality, concludes the operation should not be done except in the early stages. Chevalier believes that surgical interference is not warranted in children. In England the operation is not looked upon with favor. Fenwick states (*Statistics of Recovery after Nephrectomies*), "The sarcomata of children hardly justify operation."

The judgment of American surgeons favors operation. Holt, Jacobi, Abbe, and most American authorities advise operation. The best results are obtained, as might be expected, in early operations before cachexia develops and before damage is done to surrounding organs. From a study of 74 operative cases Walker gives an immediate mortality of 38.25 per cent., an ultimate mortality from 74.32 to 94.53 per cent., and 5.47 per cent. of cures. In my opinion, Walker well expresses the

situation: "Although the cures are very few, still, in consideration of the invariably fatal termination of this malady without interference I should unhesitatingly advise operation, for it offers the only hope and, at worst, it means only an accelerated death."

### TUBERCULOSIS OF THE KIDNEY.

Although over 70 cases of primary tuberculosis of the kidney in children are referred to in the literature, in many of these the records are so imperfect that one is led to doubt the exact location of the primary lesion. Well-studied cases, however, have been reported, the age ranging from eight months to thirteen years. Secondary tuberculous nodules of the kidneys are quite common in general tuberculosis, and may also be found in the upward spread of a genitourinary tuberculous, the primary focus of which is in the testicle. This method of infection is rare in children.

**Pathology.**—Miliary tubercles are generally found scattered throughout the kidney in general miliary tuberculosis, and in chronic pyelitis small nodules are common. The upper part of the ureter and the pelvis of the kidney are often involved. Perirenal abscess also may be tuberculous. The process differs in no essential respect from that of tuberculous foci in general.

**Symptoms.**—There will be progressive emaciation with fever fluctuating, usually within moderate limits. Vesical irritation may occur when the urine contains much pus. A decided albuminuria may be present. Hematuria is frequent. The sediment usually contains much pus, blood corpuscles, and casts. The tubercle bacillus may be demonstrated occurring either singly or in clumps. Tumor can be demonstrated in about one-half the cases. When the disease is unilateral, the ureter on the affected side may become blocked, and for a few days normal urine may be passed from the healthy kidney.

**Diagnosis and Prognosis.**—The disease is recognized by the symptoms above noted and the demonstration of the tubercle bacillus. Residual elements in the urine and anything more than a trace of albumin can be accounted for by cystitis.

With early recognition and operation there is some hope for recovery. Without operation the prognosis is uniformly bad. Death usually results from general tuberculosis. Uremia may occur, or perforation into the peritoneal cavity may terminate the case.

**Treatment.**—This is purely surgical. Vesical irritation may be relieved by the use of urotropin and plenty of water. Injections of tuberculin, under careful management, are now advised. In general, curative treatment must be surgical. The sustaining treatment necessary for all patients with tuberculosis must be carried out.

**HYDRONEPHROSIS.**

Hydronephrosis is a not uncommon congenital condition of the kidney. Occasionally it is associated with other malformations.

**Etiology.**—Hydronephrosis results from mechanical obstruction of the outflow of the urine at some point along the urinary tract, although it is not always possible to demonstrate it on autopsy.

Among the conditions in the ureter causing obstruction are: twisting, contraction, or obliteration; a pinhole vesical orifice, an acute angular junction with the pelvis of the kidney, cysts in the mucous membrane, and impacted calculus, with or without ulceration and cicatrization. While most frequently in the ureter, the obstruction may be in the bladder, urethra or prepuce.

**Pathology.**—The pelvis of the kidney is dilated into a sphenoidal sac, the calices widened and forming pockets. The cortical and medullary substance of the kidney is compressed and often destroyed, leaving in the place of the kidney only a loculated cyst. The hydronephrotic kidney may be smaller than the normal kidney, or it may form a tumor filling the greater part of the abdominal cavity.

The contained fluid may be pale and clear or dark, brownish, and colloid in consistence. It contains sodium chloride, urea and urates and epithelial cells. Urea is often present in but a very small quantity.

The ureters are elongated, sacculated, and dilated, and when the obstruction is in the lower part of the urinary tract the bladder is hypertrophied. The remaining kidney tissue is often the seat of a chronic diffuse inflammation.

**Symptomatology.**—Hydronephrosis may be unilateral or bilateral. When unilateral and the other kidney is normal, there will be no symptoms unless the hydronephrosis reaches a sufficient size to form an abdominal tumor. According to Holt, this is most frequently noted between the third and eleventh years. Nephritis of one or both kidneys is not infrequently a complication.

When bilateral, chronic nephritis or pyelitis, or both, supervenes in the early months of life, the general and local symptoms of that condition are present, and usually determine a fatal result before the development of a tumor. The hydronephrosis can in these cases only be suspected and it is commonly overlooked.

**Prognosis.**—Infants with a double hydronephrosis live but a few months, dying of nephritis, marasmus, or some other condition dependent upon the deranged kidneys. With a single hydronephrosis the outlook is gloomy, but with one normal kidney surgical interference may bring about a recovery.

**Treatment.**—This is surgical, but some benefit may be had from the administration of urotropin.

**CYSTIC DEGENERATION OF THE KIDNEY.**

This condition is occasionally met with in infants dying in the first year of life. There are usually no symptoms referable to the kidneys.

FIG. 167



Misplacement of left kidney in female infant.

The kidneys are found to be small and the renal tissue converted into large numbers of conglomerated cysts of varying sizes. The glandular structure is more or less replaced by loose connective tissue.



**MALPOSITION OF THE KIDNEY.**

Malposition of the kidney is a rare condition. In about 25 per cent. of the cases the left kidney is the one that is displaced. The displacement is usually downward and the kidney may be found lying in the hollow of the sacrum.

There are no symptoms referable to the kidney and the condition is not necessarily of clinical importance. Fig. 167 shows the left kidney displaced downward lying behind and a little above the uterus. The infant died of an acute bowel infection.

**MOVABLE KIDNEY.**

The attention given in the last few years to movable kidney has revealed its frequency in the adult and its occasional occurrence in the child. A number of isolated cases have been reported. Comby reported 18 cases, 2 being under the age of three months and 6 between one and ten years. 16 of the cases were girls and 2 were boys, about the proportion met with in adult life.

**Etiology.**—The great predominance of the condition in the female, both child and adult, would tend to show that the conformation of the female abdomen predisposes to it. Chronic dyspepsia with gastro-ectasia is an almost uniformly present condition and probably bears an etiological relationship. Many writers believe the condition congenital, dependent upon too long a pedicle.

**Symptomatology.**—In many cases the condition is latent and gives rise to no symptoms directly referable to the kidney. Paroxysmal pain in the upper or lower quadrant of the abdomen is sometimes present, appearing particularly after muscular exertion or fatigue.

The palpable kidney is usually sensitive. Attacks of nausea and vomiting are frequent, but may be dependent upon the associated gastric disease. Rarely twisting of the ureter and occlusion, with the formation of hydronephrosis, has been observed.

**Diagnosis.**—The diagnosis is often difficult, particularly in the young infant. The presence of a hard, round, movable tumor in the upper quadrant of the abdomen and replaceable under the ribs is characteristic. Appendicitis, perinephritis, stone, and renal growths of the kidney have to be differentiated.

The following is an example of the condition: Girl, aged three and one-half years. She was seen in consultation for chronic intestinal indigestion, with impaired nutrition. From infancy the child was subject to frequent attacks of vomiting, with abdominal pain and sometimes diarrhea. The attacks came without apparent dietetic cause. At the time of the consultation the child was having one of these disturbances. With no history of dietetic error, she had vomited the night before and had pain and diarrhea. The abdomen was distended and tender. The

liver was enlarged, the lower border one and one-half inches below the ribs. The kidney was palpable below the umbilical line, was markedly sensitive, and readily slipped back under the ribs. The case was not under observation long enough to determine whether the movable kidney was a coincidence of the intestinal catarrh or bore an etiological relation to it, although under a carefully regulated diet for two weeks the symptoms of indigestion disappeared.

**Treatment.**—Medicinal treatment is of limited value. A properly fitting bandage, while not holding the kidney in position, prevents by general pressure its too free excursion from its bed. Most important is the proper treatment of the associated digestive disturbance and the relief of the dilated stomach. During periods of unusual pain, rest in bed may be essential and a properly fitting bandage may be tried.

In severe cases surgical treatment is advisable.

# SECTION X.

## DISEASES OF THE BLOOD, LYMPHATIC SYSTEM AND GLANDS.

By JOHN RUHRÄH, M.D.

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### CHAPTER XXXII.

THE BLOOD—ANEMIA—CHLOROSIS—LEUKEMIA—PURPURA—  
HEMOPHILIA.

#### THE BLOOD.

THE present state of knowledge of the blood conditions of infants and young children is very incomplete. Much remains to be learned and much of what is known is obscure and difficult of interpretation. For the general purposes of diagnosis and prognosis, however, the results of blood examinations are in the main satisfactory. Every practitioner should be equipped to make routine blood examinations when required. This includes counting the red and white blood cells, an estimation of the hemoglobin, and a microscopic study of fresh or dried and stained slides.

**Differential Counting.**—It is often desirable to determine the percentage of the various kinds of leukocytes present. To do this about five hundred leukocytes should be counted. Counting is best done by using a mechanical stage, but this is not essential. It is best to start at one corner of the slide and move across one field at a time, noting the number and kind of leukocytes present. Having reached the other end of the specimen, a field lower down is counted and the reverse section taken, thus following out a serpentine course until the entire slide has been gone over (Figs. 168, 169, 170 and 171).

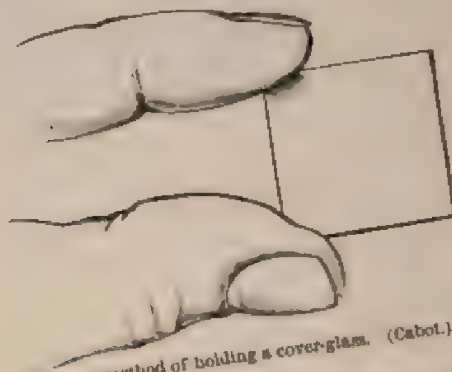
**Red Blood Cells.**—The number of these, the relative hemoglobin content, the size, shape, and staining reaction are important. It should be noted whether any abnormal cells are present. The size is on an average  $7.5\mu$ . In disease they may be very small,  $4\mu$  to  $1\mu$ , so-called *poikilocytes*. These are seen in some cases of chlorosis and in severe acute and chronic anemias. The size may be increased to  $10\mu$  or  $20\mu$ . These are called *megalocytes*. They are seen in severe anemias, usually

# 810 DISEASES OF BLOOD, LYMPHATIC SYSTEM AND GLANDS

of some duration. They are supposed, by some, to indicate an effort at regeneration of the blood.

The cells may be misshapen (poikilocytosis), and this is seen in severe grades of anemia. Care should be taken not to mistake artefacts for poikilocytes.

FIG. 168



Proper method of holding a cover-glass. (Cabot.)

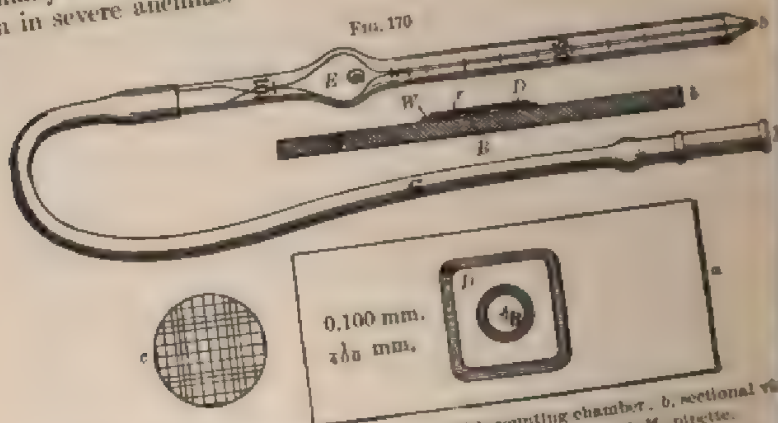
FIG. 169



Illustrating the position of cover-glass during the spreading of blood films. (Cabot.)

When stained with hematoxylin and eosin or Ehrlich's tricolor dye the red blood cells sometimes exhibit curious staining reactions. They stain a brownish color. This is known as polychromasia. It is seen normally in fetal blood and in bone-marrow cells. Pathologically it is seen in severe anemias.

FIG. 170



Thomas-Zeiss blood-counting apparatus. a, slide with counting chamber; b, sectional slide with counting chamber; c, ruled disc for counting. S. M. pipette.

Grawitz has described a granular degeneration of the red blood in pernicious anemia when blue granules or arens are seen. Nucleated red cells are seen in fetal life, in premature infants, and in some cases in adult life. They disappear after a short time in full-term life. This is pathological.



There are two varieties: 1. *Normoblasts*, which are the size of a normal red blood cell, but which have a dark-staining nucleus. These are seen in mild and severe anemia, chlorosis, leukemia, etc. In children they may be seen in bone-marrow disease and even in severe leukocytosis. 2. *Megaloblasts, giantoblasts*, are very large cells,  $10\mu$  to  $20\mu$  in diameter, showing polychromasia and several different kinds of nuclei. They are observed in young infants, as mentioned above. In moderate anemias some may be seen. If present in a severe anemia in great numbers a diagnosis of pernicious anemia should be made. This is usually a primary pernicious anemia, but great numbers may occur in the severe or pernicious type of secondary anemia.

The number of red blood cells in infancy is somewhat above that of later life. At birth the average is from 4,500,000 to 6,500,000 per c.mm. This falls during the first weeks, and for the first year of life an average of 5,500,000 may be given for healthy infants and 5,000,000 as an average for childhood.

The hemoglobin is also high at birth, usually above 100 on von Fleischl's scale. It sinks to about 100 by the second week and falls until about the third month. From this time to the second year it is low, ranging between 60 and 80. After the second year it increases until about puberty. It should be borne in mind that the hemoglobin is extremely variable in childhood.

The specific gravity, alkalescence, and other things often mentioned have as yet no great practical interest.

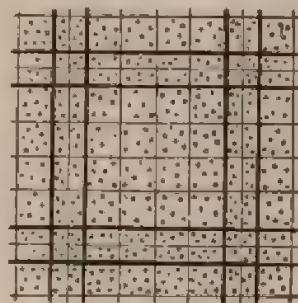
**The White Blood Cells.**—These are of particular interest and are of various kinds. Ehrlich's classification is as follows:

*Lymphocytes: Small Mononuclear Leukocytes.*—These are small cells about the size of a red blood corpuscle. The nucleus occupies the greater portion of the cell. The nucleus stains well with basic dyes, but not as deeply as the narrow rim of protoplasm which surrounds it (basophile).

*Large Mononuclear Leukocytes and Transitional Forms.*—These are large cells two or three times larger than the preceding. The nucleus is oval, usually not quite in the centre, and stains with basic dyes. It does not stain as deeply as the nucleus, but is always much darker than the protoplasm which surrounds it. The protoplasm is clear, contains no granules, and forms a considerable portion of the cells (basophilic).

The transitional forms resemble slightly the following in that the nucleus is more or less irregular in shape and stains more deeply than in the simple large mononuclear form. The protoplasm may contain a few granules which stain only with neutral dye (hence neutrophilic granules).

FIG. 171



Appearance of blood in the Thoma-Zenker cells.

## CELLS OF BLOOD, LYMPHATIC SYSTEM AND GLANDS

**Polymorphonuclear neutrophilic leucocytes.** called generally, for convenience, polynuclears. These are slightly smaller than the preceding. The nucleus consists of several pieces joined by narrow strips of protoplasm. The nucleus stains deeply with basic dyes. The protoplasm stains with acid dyes and instead of being clear is filled with numerous small granules which stain only with neutral dyes.

**Eosinophiles** are in general appearance like the preceding except the nucleus has usually but two parts. The granules are larger and stain with acid dyes (eosin for example).

**Mononuclear cells** are seen only occasionally. They resemble the polynuclears in general appearance but the nucleus may be mononuclear or polynuclear and the granules stain only with basic dyes.

**Normal White Cells. Myelocytes.**—These are large cells which normally, like the nucleated red cells, belong in the bone-marrow. They are occasionally found in the blood in certain diseases, as diphtheria, starvation and various toxemias, as well as in splenomegaly.

When Rich's tricolor dye they are seen as large round or nearly round cells. They have a large nucleus which takes but a pale stain, and the surrounding protoplasm is filled with neutrophilic granules. Sometimes the granules may be more or less basophilic. The size is usually larger than any of the cells described above, but the diagnosis of a myelocyte is made on the staining reaction rather than mere size, they may be small.

**Basophilic Myelocytes.**—These are like the preceding except the granules are stained by acid dyes (oxypink).

In addition to the above other cell forms are seen occasionally, most important of which are degenerated leucocytes. These are leucocytes staining feebly or intensely, usually without a nucleus, or with vacuoles. Non-granular myelocytes may also be seen in any very severe anemia.

**Blood plates** are found in normal blood, but are usually overlooked. They are generally seen clumped together. They are half the size of a red blood cell, are colorless, and have no amoeboid movement. Their clinical significance, if they have any, is not known.

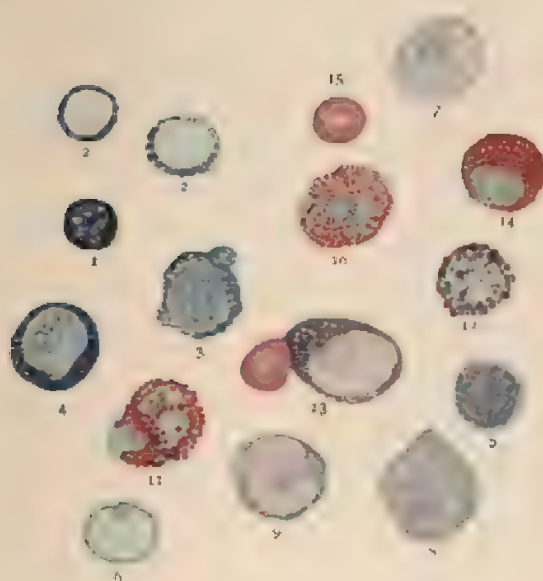
**Blood Dust.**—In fresh blood there are seen numerous highly refractive, actively dancing bodies. These are supposed to be the granules of free iron from the eosinophiles. They should not be mistaken for malarial parasites.

FREQUENCY OF VARIOUS FORMS OF LEUCOCYTES

	Infancy.	Adult.
Lymphocytes	40-60	20-30 per cent.
Large mononuclears	—	4-5
Polymorphs	25-40	40-55
Eosinophiles	2-4	10-14
Mononuclears	—	1-6-12

The total number of leucocytes in the blood in infancy is somewhat greater than adults. They are highest at birth, from 12,000 to 25,000. They fall rapidly during the first few days and reach an average between 9,000 and 14,000. During childhood the average is still lower, from 6,000

## PLATE XXI.



Note the size of the various leukocytes, as compared with the red corpuscles at 15. Figs. 1, 2, and 6 represent the most common forms of the small type of lymphocytes; 3 and 5 belong to the same group, but are manifestly atypical; 3 shows the knob-like projections; 4 represents the large type of the lymphocyte, and shows the vacuolated appearance of the protoplasm, which is so commonly seen. The metachromatism of the protoplasm, however, does not appear here as in nature. 7 and 8 are representatives of the large variety of mononuclear leukocytes; 9 may be classed as a transition form, which is as yet devoid of granules; 13 represents a neutrophilic myelocyte, 11 an eosinophilic myelocyte, 10 a neutrophilic polynuclear leukocyte, 12 a typical basophilic leukocyte.

The preparations were stained with the eosinate of methylene blue and drawn to scale (Hausch & Lomb, eye-piece 4 inch, objective  $\frac{1}{16}$ ). (Simon.)





PLATE XXII.



Tallquist's Color Scale for Estimating Hemoglobin. (Musser.)



5,000. Further study is needed to determine the number and variety of various forms of leukocytes at different ages. (See Plate XXI.)

**The Significance of Blood Changes.**—Red blood cells are diminished in primary and secondary anemias, are about normal in chlorosis, and are increased in cyanosis. They are increased in high altitudes and in sea sickness. This may be taken advantage of in the treatment of anemia.

**Hemoglobin** is diminished in all forms of anemia. In chlorosis and secondary anemia the corpuscle contains less than normal. In pernicious anemia it may contain more than normal, but the total quantity is diminished because the total number of red cells is also diminished.

In the cyanosis of congenital heart disease there is a concentration of blood. There is an increase in the number of red blood cells, from 4,000 to 12,000,000 being noted. There is increased specific gravity and increased hemoglobin. In some cases there may be an increase of about a thousand per millimetre in the leukocytes. (See Plate XXII.)

**Lymphocytes** are normally much more abundant than in adults. Many of the so-called cases of lymphocytosis are only the normal lymphocytes of early life. In many severe diseases, as in gastroenteritis, the blood of children tends to revert to the infantile type and there is an increase in the number of lymphocytes. These cells are increased in whooping-cough, rickets, scurvy, and especially in hereditary syphilis. The greatest increase is in lymphatic leukemia. The increase must be relative and absolute before making the diagnosis of lymphocytosis. If the polynuclear neutrophils are diminished there may be a relative increase in the lymphocytes. In syphilis where there is doubt as to the diagnosis, an increase in the lymphocytes, especially if coupled with an increase in the eosinophiles, points to syphilis. Cabot has suggested that the number of lymphocytes in the blood of a child might be used as a measure of its development, excluding causes for leukocytosis, the standard being the normal percentage for a child of the given age.

**Leukocytosis.**—This may be physiological or pathological. Physiological leukocytosis of all kinds is exaggerated in infancy and childhood. Leukocytosis of the newborn has been considered. Fasting lowers the number of leukocytes, while taking food increases them. After a meal 30,000 leukocytes may be counted. This increase begins about an hour after the meal and lasts several hours. There is leukocytosis after exercise, massage, and cold baths. A leukocytosis is frequently observed just before death. This is called *agonal leukocytosis*.

Pathological leukocytosis, affecting chiefly the polynuclear neutrophils, occurs in numerous conditions, as in malignant tumors, in anemias, owing to various drugs or experimental procedures, after hemorrhages, and especially in inflammatory conditions. Of

importance are the diseases where there is pus formation, as in abscesses, peritonitis, osteomyelitis, as well as septicemia and pyemia. In pyemia it is of some diagnostic value and a sudden increase in leukocytes late in a pneumonia or during convalescence frequently indicates an empyema. It is useful in differentiating a catarrhal from a suppurative appendicitis. Too much stress should not be laid on the

importance of leukocytosis in surgical diseases of very young children. The subject needs further investigation.

In minor infections the leukocytosis is of a mild grade, in moderate or severe inflammations where the resistance is good it is marked, but in very severe infections there may be no leukocytosis.

In *pneumonia* there is a reduction in the hemoglobin and red blood cells, and in all but exceptionally mild or very severe cases a marked leukocytosis. In children this is especially marked, 50,000 being frequently noted. The absence of leukocytosis in severe cases means a bad prognosis. In obscure or in centrally situated pneumonias the leukocytosis may be of considerable diagnostic value. The eosinophiles are diminished or absent and their reappearance is taken to mean that the acme of the disease has been passed.

In *diphtheria* there is a normal red blood count which falls after the third or fourth day. The hemoglobin also diminishes. The return to normal is slow. In cases treated with antitoxin the loss of red blood cells and of hemoglobin is not so great. Leukocytosis is present in nearly all instances. It may be absent in very mild or very severe cases. Engel found myelocytes in very severe cases. Where they exceeded 2 per cent. the patients died.

*Scarlet fever* results in a diminution of the red blood cells and of hemoglobin. The leukocytosis varies with the intensity of the disease. It reaches its height one or two days after the appearance of the rash and falls gradually, persisting after the eruption. The eosinophiles are said to increase after two or three days and reach a maximum of from 8 to 15 per cent. in two or three weeks. They then fall gradually, reaching normal about the sixth week. According to Neusser the eosinophiles are increased in favorable cases and decreased in the unfavorable ones. In differentiating measles and scarlatina, a leukocytosis by the third day points to scarlatina.

*Whooping-cough* shows a marked and early leukocytosis. This appears in the catarrhal stage and disappears slowly with complete convalescence. The number averages 25,000 to 30,000, and it is pronounced in children under four years of age, about one-half the white cells in these cases being lymphocytes. This is of value in differentiating whooping-cough from spasmodic cough caused by pressure of tuberculous bronchial or mediastinal lymph nodes.

*Varicella*.—Few observations have been made. Engel has reported moderate polynuclear neutrophilic leukocytosis with eosinophilia after healing.

*Vaccinia*.—Leukocytosis begins on the third or fourth day after inoculation and then falls to the seventh or eighth day, when the leukocytes may even fall below normal. There is a secondary leukocytosis on the tenth or twelfth day, lasting from two to six days (Sabotka).

*Acute articular rheumatism* shows an anemia with leukocytosis varying in a general way with the severity of the disease. It has no diagnostic value, however, as the same is found in other arthritides.

*Meningitis*.—Septic meningitis has a leukocytosis. Cerebrospinal fever has it in about two-thirds of the cases. In tuberculous meningitis



there is usually no leukocytosis, although there are exceptions to this. For the first two leukocytosis is often of value in excluding coma from other causes or typhoid resembling meningitis. There is leukocytosis in brain abscess.

In quite a number of diseases there is no leukocytosis unless there are complications. It is important to bear these in mind. The most prominent are tuberculosis in its various forms, typhoid fever, malaria, mumps, measles, and German measles. Influenza is said not to have leukocytosis in most cases.

*Leucopenia*, or a diminution of the white blood cells, may be present at times in any of the diseases just mentioned, in malnutrition, usually in very severe anemias, and in leukemia when complicated by an infectious disease, and in a few other conditions.

*Eosinophilia*, or an increase in the number of eosinophiles, is found in a very large number of conditions and is of some diagnostic and prognostic value. Among the conditions where it is found are: infection of the body with most of the animal parasites, as in trichinosis, ankylostomiasis, and the various forms of intestinal worms, oxyuris, ascaris, and the tapeworm; in malignant tumors, in many other diseases both acute and chronic; especially pemphigus and urticaria; in purpura and hemorrhagic exudate; in diseases where the bone-marrow is affected; in leukemia, in scarlet fever, and sometimes in rheumatism, and after fevers. The presence of eosinophilia shows active regeneration of the blood and is looked upon as a favorable sign in severe anemias following hemorrhage. It is also supposed to mean a good prognosis in scarlet fever and chlorosis.

*Mast cells*, according to Ewing, are seen with greater frequency in patients from the lower classes than in the well-to-do. They may be increased in some cases of leukemia and have been seen in other diseases.

*Myelocytes* are seen under several conditions. They are present in large numbers in most cases of leukemia. They may be seen, however, in small numbers in severe anemias of any form, in the leukocytosis of some infections (diphtheria) and after any severe blood disturbance, as uremia, asphyxia, and the like.

#### ANEMIA.

The anemias of infancy and children are deserving of further study. Our knowledge is as yet chaotic and fragmentary. Several things must be borne constantly in mind. The age of the child and the blood condition which is normal to that age are important. If a child is backward in development its blood corresponds to the age of a child which it resembles. There is a tendency to revert to the embryonic type or to the type of the younger child. Normoblasts may be seen in early infancy and have no especial significance. Leukocytosis may be present in a severe anemia. In infants a large spleen may be seen with any

form of anemia. "All the signs by which disease is shown by the blood of adults are exaggerated in children." (Cabot.)

**Simple or Secondary Anemia.**—Simple or secondary anemia is that which is due to some known cause in contradistinction to primary or the so-called pernicious anemia when the cause is unknown and where a certain blood condition exists. Some of the anemias designated as pernicious might be classed as secondary now, as they have been found due to certain intestinal parasites. As the blood changes are the same as in the pernicious anemia, they are considered with that disease. Simple anemia may be of any grade from the most trifling to the most severe, and even fatal forms may be met with. The anemia may start as a simple anemia and later take on a pernicious character.

**Etiology.**—Simple anemia is exceedingly common in infancy and childhood. Owing to the demands on the organism, anything which interferes with the proper nutrition is liable to cause anemia. Disease or excessive weakness of the mother during pregnancy may be the cause of a weak child which soon becomes anemic, due to lack of power to form sufficient blood. Children who have insufficient food, light, and air are always anemic. Hemorrhage in infancy and childhood may be followed by severe anemia. The very young are much more affected by hemorrhage than are adults. The administration of certain drugs may cause anemia, among them mercury and chlorate of potassium are in common use. Anemia may follow almost any disease.

It may be due to toxins which are produced in the body, or it may be owing to the fever, to malarial or other parasites.

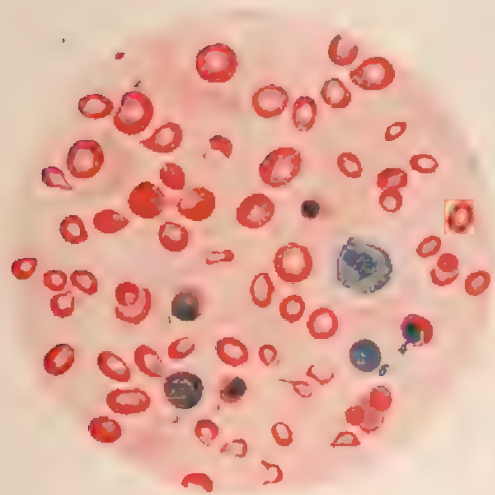
**Classification.** There have been numerous attempts to classify the secondary anemias. As yet all classifications are arbitrary and do not seem worth while. The presence or absence of an enlarged spleen, of leukocytosis, and of the severity of the disease are the usual basis for the division.

**Pathology.**—The blood condition differs with the severity of the disease. The differences seem to be one of degree, but it should be borne in mind that in infancy any severe anemia tends to bring the blood back nearer to the embryonic type or to the type of a younger child.

The hemoglobin is lowered; in severe cases it may go to 30 or under. The specific gravity is also lowered. The red blood cells are diminished in number, varying from normal to 1,500,000 or even lower than that. There is poikilocytosis and some difference in size of the cells. The cells are usually undersized (microcytes), but megalocytes may be met with. There may be polychromasia in severe forms. Nucleated red blood cells are seen in varying numbers. Normoblasts are present in the average cases and megaloblasts may be seen in the severe case. There may or may not be leukocytosis. This has been considered under that heading. There is more likelihood of leukocytosis with secondary anemia in infancy and childhood than in adult life, and the number of red cells is usually much lower. (See Plate XXIII.)

**Symptomatology.**—The symptoms vary with the intensity of the disease. In the milder cases there is pallor of the skin and mucous

PLATE XXIII



Blood from Case of Secondary Anemia. (Musser.)

- |                 |                                  |
|-----------------|----------------------------------|
| 1. Poikilocytes | 3 and 6. Lymphocytes             |
| 2. Macrocytes   | 4. Nucleated Red Blood Corpuscle |
|                 | 5. Polynuclear Leukocytes        |

(Oc. 1, ch. 1, immersion) Drawn by J. D. Z. Chase





membranes, languor, loss of appetite and, as a rule, some digestive impairment. In infants and children there may be marked irritability and peevishness. This in a previously good-natured child is very suggestive.

In the severe cases the pallor is extreme and if it is a case of long standing there may be a slight yellowish tint in the skin. There may be slight edema. The tongue is usually coated, the appetite lost, the nutrition poor, and either constipation or diarrhea is liable to be present. The circulation is poor. If old enough the child complains of being chilled on the slightest exposure. The hands and feet feel cold. The heart sounds are weak, and there may be a dilated heart with numerous murmurs, and an enlargement of the area of dulness. Of greater frequency are hemic murmurs heard over the base of the heart and over the veins in the neck. The respiration is more rapid than in health and breathlessness results from slight exertion. These children tire easily from any effort. The patients are irritable and capricious; headaches are frequent, and indefinite pains are complained of. The sleep is poor and the patient may be very wakeful. There are liable to attacks of fainting. Enuresis may be seen, which usually disappears when the child regains its health. Emaciation is the rule, but some infants do not grow thin. Anemic children catch cold easily and are prone to catarrhal conditions of all kinds. There may be hemorrhages from the nose or other mucous membranes.

The *spleen* is enlarged in some cases, especially where the predisposing cause is usually accompanied by an enlarged spleen. The liver may also be enlarged.

**Diagnosis.**—This is, as a rule, easy. The blood changes and the persistence of the cause are sufficient. If very severe with megalocytes, galoblasts, and polychromasia it may be impossible to distinguish simple or secondary anemia from the primary or pernicious form, unless the history of the case is known.

If the spleen is enlarged and leukocytosis present it may bring to mind lymphatic leukemia or the pseudoleukemia of infants. The resemblance may be striking in either instance. One should not be too hasty in coming to a conclusion in the former case if there is the history of one of the causes of lymphatic leukemia. The leukemia is progressive, while the anemia is apt to be temporary and to improve.

In pseudoleukemia (see p. 824) the spleen is usually larger and leucocytes are found in considerable numbers.

**Prognosis.**—Prognosis depends on the cause. If it can be ascertained and removed and the blood condition is neither very severe nor of long duration the outlook is good. If the anemia is severe the prognosis must be guarded. Monti states that the cases with leukocytosis are more prone to develop into severe anemias than those without it.

If the hemoglobin is reduced to below thirty or the red blood cells 2,000,000 or nearly that, the case may be regarded as very severe. The same applies to the presence of many megalocytes, megaloblasts, to much polychromasia. A high color index is also a bad sign.

**Pernicious Anemia.**—Biermer called progressive pernicious anemia those cases where there was no assignable cause and where there was a gradual progressive increase in the severity until death took place. We now call pernicious anemia those cases which have a definite blood picture which is given below. It may rarely happen that a case recovers. In children probably three-fourths of the cases have either an assignable cause or they are cases which have developed from secondary anemia, the blood picture changing from one to the other.

**Etiology.**—It is rare in infants and children. Monti and Berggren give 16 cases. Of these 2 occurred in sucklings, 5 from one to five years and 9 in children over five years. In 4 of these cases there was an assignable cause. Monti has stated that the severe secondary anemia of childhood with leukocytosis are liable to become pernicious. Cases of hereditary syphilis and of rickets where there is an enlarged spleen are put down as among the most frequent causes. Intestinal parasites, especially the *ankylostoma duodenale*, may be responsible for it.

**Pathology.**—The lesions found consist in severe anemia of all the organs, with extensive fatty degeneration of most of them. The heart and vessels suffer most from this, but the liver and kidneys are also affected. There are numerous small hemorrhages. There are deposits of iron found in the liver, due, according to Hunter, to the destruction of red blood cells in the liver by toxins. These toxins in some cases are supposed to come from the intestinal canal. The lymph nodes are often a dark-red color. Small hemorrhages are usually found in the various organs.

The blood changes are characteristic. The specific gravity is lowered. The hemoglobin is reduced to 40, 30, 20 or even below that. The hemoglobin contents of each cell may, however, be normal or above normal. The color index of the cell is high. The red blood cells are greatly reduced in number. There may be only 2,000,000 per c.mm. or even fewer than that. Owing to the high color index they stain well, but the coloring matter is usually taken unequally. The average diameter of the red blood cells is increased. Megalocytes are common, while microcytes are rare. There is marked poikilocytosis. The red cells may be polychromatophilic. Nucleated red blood cells are seen, both normoblasts and megaloblasts. The latter usually preponderate. Myelocytes may be seen occasionally. The red blood cells have lost their tendency to form rouleaux. The leukocytes are diminished at the expense of the polynuclear neutrophiles, which gives a relative increase in the lymphocytes. Leukocytosis due to intercurrent affections may occasionally complicate the picture. (See Plate XXIV.)

**Symptomatology.**—The symptoms are those of severe anemia. There is a waxy pallor of the skin and mucous membranes. The skin usually is a light lemon tint. There may be slight puffiness or edema, and later in the disease this may be very marked and there may be effusions into the serous cavities. There may or may not be emaciation. If there is no emaciation the extreme pallor with the apparently well-nourished appearance is almost in itself diagnostic. There is great weakness

amounting, sooner or later, to prostration. There are restlessness, disturbed sleep, and nervousness. In some there may be pain in the extremities. There is marked dyspnea on exertion. The heart is likely to become dilated and is constantly found enlarged. Hemic murmurs and those due to the dilatation are present. There is a venous hum over the larger vessels. There are digestive disturbances. As the disease progresses there are hemorrhages from the mucous membranes and under the skin. The urine is small in amount, of low specific gravity, and contains no albumin. As a rule, there are no appreciable clinical changes in the liver, spleen, or lymph nodes.

**Diagnosis.**—This may be difficult at the start or under certain conditions, and impossible without a blood examination. Although the general clinical picture of a severe anemia is sufficiently clear, yet the prognosis depends often on the nature of the blood change. This is particularly true of a child where an anemia may be very severe as far as general symptoms go, but which still shows the characteristics of a secondary anemia. If the cause can be removed and the child managed properly recovery may be rapid. If the blood change is that of pernicious anemia, however, the outlook is bad.

Eosinophilia in a severe anemia may point out a cause, as it is seen when there are intestinal parasites. These should be looked for in all cases and especially when the eosinophiles are increased.

From other blood conditions the diagnostic points are as follows: Severe *chlorosis* may clinically suggest pernicious anemia owing to the well-nourished condition, the tinting of the skin, and the striking pallor. The number of red blood cells is rarely anything like as low in chlorosis, in which, as a rule, it is not far from normal. The color index of the cells is very different. In chlorosis it is low. Many of the cells look like colorless shadows. In pernicious anemia it is high and the cells are dark. They are also liable to be larger. Megaloblasts have been noted in chlorosis, but are never a feature of the disease.

In *secondary anemia* the number of red blood cells is not so low, as a rule. There may be a leukocytosis from the original cause. The color index of the red cells, their increased size, and the presence of megaloblasts in abundance are the greatest helps.

In *leukemia* the diagnosis may not be as easy as it would seem, especially in infancy, where there may be a leukocytosis and an enlarged spleen in any anemia. The red cells are more liable to be reduced from leukemia in infants. The large number of myelocytes in the leukemic blood is the most distinguishing feature.

The *pseudoleukemia* of infants (von Jaksch) is discussed under that disease (p. 822).

**Prognosis.**—The course of the disease is progressively downward, but there are remissions where the blood state and the general condition may improve. There may be fever with the exacerbations. The average case is more rapid in the child than in adults and the height of the disease is reached in six or eight weeks. The disease usually lasts several months before death takes place. Very rarely it may

color index; (c) increase in size of red cells; (d) degenerated red cells; (e) numerous megaloblasts; (f) few or no normoblasts; (g) numerous normoblasts.

**LESS SEVERE (SLOWER COURSE).—**(a) remissions; (b) normal color index; (c) normal size or small cells; (d) no degenerated red cells; (e) numerous normoblasts; (f) few megaloblasts; (g) no polymorphonuclear cells.

**Treatment.**—Treatment of pernicious anemia is discussed under that heading.

### CHLOROSIS.

Chlorosis is a primary anemia in which there is a diminution of hemoglobin without any marked diminution of the number of blood cells, except in very severe cases. It occurs most frequently at puberty and there is a characteristic greenish-yellow color of the skin which has led to the popular name of "green sickness."

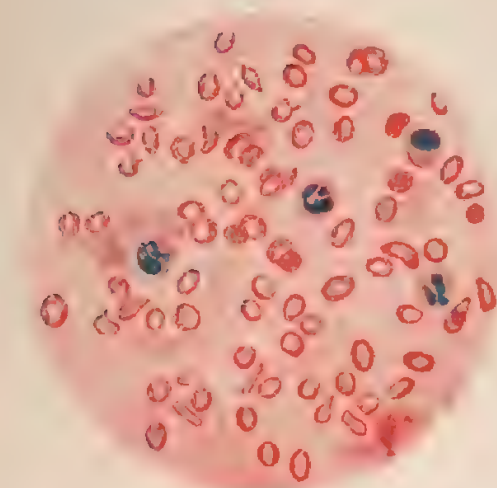
**Etiology.**—The exact cause is unknown. It occurs, as a rule, and is almost always seen in girls. Occasional cases may be seen in boys. It is more frequent in brunettes than in blondes. Previous ill health and more especially bad hygiene are common causes. The majority of the cases are seen in girls who have been brought up in a confined environment, with lack of fresh air, sunshine and light, of exercise in the open air, and of proper food. Overcrowding and overwork make it especially common in factory and shop girls. Marked psychical disturbance is also a cause. Virchow put down as a cause the congenital narrowing of the aorta and bloodvessels and a small-sized heart. This could not be the case as most cases recover perfectly. These changes are, however, characteristic of the status lymphaticus, in which there is frequently a diminution of the blood.

**Pathology.**—Cases rarely come to autopsy. Those that do are usually found to be complicated with tuberculosis, when of the



# PLATE XXIV.

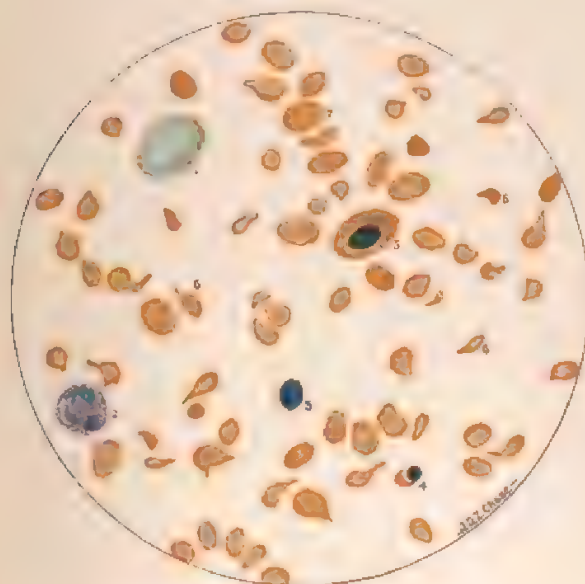
FIG. 1.



Blood from Case of Chlorosis, showing Slight Staining of the Red Blood Corpuscles and Presence of Mononuclear Leukocytes. (Musser.)

(Oil-Immersion.) Drawn by F. D. Z. Chase.

FIG. 2.



Pernicious Anemia. (Musser.)

- |   |                       |
|---|-----------------------|
| 1. Large Mononuclear Lymphocyte.              | 5. Small Lymphocyte.  |
| 2. Polymorphonuclear Leukocyte or Neutrophil. | 6. Polkilocyte.       |
| 3. Microblast.                                |                       |
| 4. Nucleated Red Corpuscle.                   |                       |
|   | Normal Red Corpuscle. |

-  
1  
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1

that in severe cases the number of red cells may fall to two or three million per cubic millimetre. The color index of the cell is, however, lowered. The size and shape of the cell are altered and these are more marked in the severe than in the mild cases. Some of the cells may have so little hemoglobin as to look like faint round shadows. Other cells are somewhat smaller than normal and there may be poikilocytosis. Normoblasts may be seen sometimes in the severe cases. The leukocytes are, as a rule, normal, but there may occasionally be a slight leukocytosis. (See Plate XXIV.)

**Symptomatology.**—The symptoms are much like those of a simple anemia, but there is no emaciation, the well-nourished appearance being in striking contrast to the pallor and tint of the skin. The skin has a greenish-yellow color and there may be patches of darker pigmentation. There is frequently slight puffiness or edema present. There is shortness of breath on exertion. Palpitation of the heart is common and there is a rapid, weak pulse. The heart is often dilated if there has been much severe exertion. This is usually seen in the right ventricle, but the left may also be affected. There may be some hypertrophy of the left ventricle. Hemic murmurs are heard over the base of the heart and a venous hum over the large vessels in the neck. There is a coated tongue, a capricious appetite, and unusual longings for all sorts of strange articles of diet. After eating there is often discomfort or indigestion. Pain in the region of the stomach is a frequent complaint. Hyperacidity may be present and gastric ulcer may complicate the disease. Constipation is the rule. There is generally amenorrhea in older girls. Slight albuminuria may be present. There may sometimes be a little fever.

The patient is nervous, fretful, and irritable. Attacks of crying from slight causes are not uncommon. There may be hysterical attacks. Vertigo is of frequent occurrence and, if not that, the patient complains of attacks of faintness.

The duration is that of a chronic condition. The cases last months or even a year or more. The course varies. There may be periods where the condition is reasonably good followed by relapses.

**Diagnosis.**—Diagnosis is easy. The disease can usually be recognized at a glance. The blood examination settles the question. Care should be taken to recognize those cases associated with the status lymphaticus.

**Prognosis.**—This is good if there are no complicating diseases.

**Treatment.**—Prophylaxis is important. Shop, factory, and school-girls should have sufficient fresh air and light and not be overcrowded. A few factories have recognized that they can save money by arranging for the health and well-being of their employes. The medicinal treatment is along the same lines as for other cases of anemia. Diet is important and iron the most efficient drug.

**PSEUDOLEUKEMIA OF INFANTS (VON JAKSCH).**

The disease described by von Jaksch, in 1889, as *Anemia Infantum Pseudoleukemica* is a rare form of anemia seen only in infants. It is characterized by a grave anemia and leukocytosis, together with enlargement of the spleen, liver, and sometimes of the lymph nodes. There has been much discussion as to whether the condition is really a separate disease, and, if it is not, whether it should be classed as a secondary anemia, as a pernicious anemia, or as a leukemia. Without entering into the discussion it may be said that it is, for the present at any rate, a good way to dispose of a certain number of puzzling anemias of early life.

**Etiology.**—The majority of cases occur between seven and twelve months of age. It has been seen somewhat earlier and also as late as three and four years.

In twenty cases collected by Monti and Bergergrün, sixteen had rickets and one hereditary syphilis. Monti is of the opinion that it may develop from severe anemias.

**Pathology.**—The spleen is large and hard. There may be thickening of the capsule; microscopically the only change is a stiple hyperplasia. The liver is enlarged in almost half the cases and is said to bear no relation to the size of the spleen. There is no infiltration of the liver with white cells as in leukemia, but there are some red and white cells found. In about half the cases there has been enlargement of the lymph nodes. Changes in the bone-marrow have been noted.

The blood condition is as follows: The specific gravity is lowered from 1.035 to 1.045. The hemoglobin is lowered, in some as much as to 30. The red blood cells are greatly diminished sometimes to less than a million, usually to between one and two millions. The red cells are frequently changed both in size and shape. There are microcytes and megalocytes as well as poikilocytosis. Nucleated red cells are present, both normoblasts and megaloblasts. The white cells are increased so that the relation of white to red cells is below 1:100. Monti gives the variations as between 1:85 to 1:15. In other words, a leukocytosis of from 20,000 to 50,000. The mononuclears and polymuclears are both increased, sometimes one and sometimes the other. The eosinophiles may be increased. Myelocytes may be present. The white cells stain differently and there may be curious appearances caused by the irregular way in which they react to the ordinary dyes.

**Symptomatology.**—The symptoms of the disease are those of a chronic anemia. There is usually, though not always, emaciation. The severe anemia causes a cachectic appearance. There is loss of appetite and digestive disturbance. The spleen is large. The liver and lymph nodes may also be enlarged. The disease may go to a certain point and then remain at a standstill. There may be periods of improvement and then periods where the patient grows worse. After dragging along for a long time the patient may die, sometimes apparently from the anemia.



sometimes from some intercurrent disease. Four cases out of Monti and Burgegrün's twenty died.

**Diagnosis.**—This may be a matter of considerable difficulty. It is on searching and weighing the differential points that one realizes on what an insecure basis the disease really stands. The symptom-complex with the blood findings taken all together are of the greatest value. Monti regards it as a sort of forerunner of *leukemia* in some cases. If the patient dies the autopsy shows a different process from leukemia. If the patient recovers it is good evidence that it was not leukemia. The leukocytes are not so numerous as in leukemia, but it must be borne in mind that under certain conditions a low leukocyte count may be found in leukemia. The percentage of myelocytes is lower in pseudoleukemia, as a rule. The liver is not so large and the lymph nodes may not be enlarged at all in some cases.

The color index is lower in pseudoleukemia as a general thing than in pernicious anemia. The number of red cells is lower in pernicious anemia. It must be borne in mind that leukocytosis may occur in any grave anemia in infancy; were it not for this the diagnosis would be easy. The general clinical picture with the larger number of myelocytes is the best means of distinguishing the two.

The fact that rickets and syphilis may both cause anemia and leukocytosis with enlarged spleen, liver and lymph nodes makes it difficult to separate secondary anemia at times from pseudoleukemia. The spleen is perhaps larger in the latter and myelocytes more in evidence. When there has been neither rickets nor syphilis the diagnosis is easier.

The nodes are larger in Hodgkin's disease and the anemia is not so severe. Should there be any doubt a section of the nodes will clear up the diagnosis.

**Treatment.**—This is the same as outlined for anemia (p. 827).

### LEUKEMIA.

This is a condition where the white blood cells are principally affected. Ehrlich has spoken of it as a "mixed leukocytosis" where all forms of white cells were increased as in contradistinction to polynuclear leukocytosis either of the neutrophilic or the eosinophilic type. There are in addition, in one of the forms, cells which normally belong in the bone-marrow—myelocytes. With these blood changes there are lesions in the spleen, bone-marrow, and in some cases in the lymph nodes. In infants it may at times be difficult to draw the line in some cases of leukocytosis and leukemia, especially of the lymphatic type.

**Etiology.**—Etiology is obscure. It is rare in infancy and childhood, but is occasionally seen. In some cases there seems to be an hereditary influence. It is more common in boys than in girls. Some of the cases are distinctly primary, no previous disease having been noted. In others congenital syphilis, rickets, malaria, simple anemia and the

various infections of childhood have been observed as preceding it. In some instances the child has had a succession of the diseases incident to early life and it is impossible to say whether there is any connection between the two or not.

There are numerous theories regarding the cause of the disease. By many it has been regarded as an infection. Löwit claims to have found a hemameba in the blood of leukemic patients which he regards as the cause. This needs confirmation.

**Pathology.** There are two types of the disease. The commonest form, where the principal changes are in the spleen and bone-marrow, is called *splenomyelogenous* or *myelogenous leukemia*. The other form is the *lymphatic*, where the lymph nodes are the principal site of disease. All the organs mentioned are involved in some cases.

The lesions found are very striking. The blood in very severe cases contains so many white cells as to approach pus in its appearance. The bone-marrow is the seat of extensive changes, consisting principally in the infiltration with lymphoid cells, which in some cases give it a greenish-yellow appearance. The spleen is enlarged. Usually the enlargement is very great, as it may take up over half of the abdominal cavity. In the more acute cases it is found to be soft, dark, and full of blood. Later it becomes harder and there may be perisplenitis. The organ is full of nodules which are made up of lymphoid cells. The Malpighian corpuscles are prominent and microscopically there is found to be a superabundance of lymphoid cells. There may be infarctions. The liver is enlarged and may contain lymphomatous nodules. In the *lymphatic* form the lymph nodes are enlarged and hard, but are usually movable. At the outset one or more groups may be affected, but later on there is liable to be a general enlargement of all of the nodes. The lymphoid tissue in the intestinal tract may be affected and also the tonsils and the lymphoid tissue about the mouth.

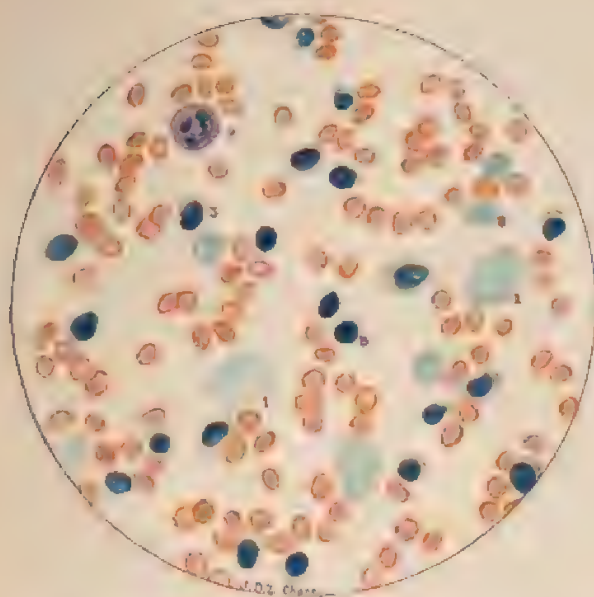
There are two types of lymphatic leukemia. In the acute there is only moderate enlargement of the spleen and a great tendency to petechia and to hemorrhages. This has been regarded as an infection. In the chronic type the spleen is very large.

The two forms, lymphatic and splenomyelogenous, get their differentiation chiefly from the blood changes. The hemoglobin is diminished. The red blood cells are usually diminished but normal in size except in very severe cases. There are normoblasts present.

In the *splenomyelogenous* form the white cells are enormously increased. The number may be as high as 500,000, while 100,000 is a common number. These consist of large numbers of myelocytes of various sizes. The polynuclear neutrophils are increased, but the percentage of them present may be decreased. The lymphocytes vary a great deal. They are increased more in some cases than in others. The large mononuclears are increased. The polynuclear eosinophiles are increased and this may be a point of some diagnostic importance, though it is not one of the especially characteristic features of the disease. The mononuclear eosinophiles are also increased. The mast

# PLATE XXV

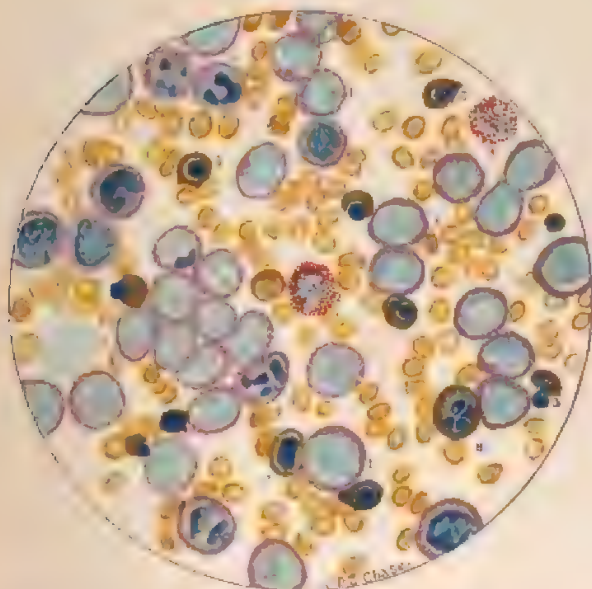
FIG. 1.



Lymphatic Leukemia. (Musser.)

1. Large Mononuclear Lymphocyte. 2. Polymorphonuclear Leukocyte or Neutrophil. 3. Small Lymphocyte, dense blue Nuclei.

FIG. 2.



Splenomegalic Leukemia. (Musser.)

FIG. 2. 1. Large Mononuclear Lymphocyte. 2. Normal Elastic Red Corpuscles, dividing or fragmenting nuclei. 3. Normal Elastic Red Corpuscles. 4. Large Mononuclear Lymphocyte. 5. Small Lymphocyte. 6. Small Lymphocyte. 7. Polymorphonuclear Leukocyte or Neutrophil. 8. Megakaryocyte.





cells may be very much increased, which is of considerable importance in diagnosis.

In the *lymphatic* form the lymphoid cells are the features of the disease. They may form as high as 80 or 90 per cent. of all white cells present. The other white cells may be increased as far as actual numbers go, but they are diminished when it comes to estimating percentages by differential counting. In some cases the blood may seem to consist almost entirely of lymphocytes and red blood cells. Myelocytes and mast cells may or may not be present. (See Plate XXV.)

Two things must be borne in mind. One is that just before death the white cells may fall to normal or below it and, secondly, there may be a return to the normal or near it when there is some intercurrent infection, as typhoid fever. After this has disappeared the leukemic condition of the blood returns.

**Symptomatology.**—The disease in infants and children is essentially the same as that seen in adult life, but it is more rapid in its course, as a rule, and it has been described as having the symptoms more exaggerated. The course of the disease is a matter of weeks or of months. The course may be very acute, and an instance is recorded where in a typical case death took place ten days after the onset in a previously healthy infant. On the other hand, it may drag on for a year or more. There is a case on record that lasted three years.

The *onset* may be sudden, but is usually very insidious. Sometimes a sudden hemorrhage calls attention to the disease which had been given no especial concern before. Ordinarily there is the gradually increasing pallor of the skin and mucous membranes. There is some digestive disturbance which is often thought to be the only trouble. There are loss of appetite, indigestion, attacks of vomiting or of diarrhea and sometimes a little fever. There is a tendency to hemorrhages. These are usually slight in the beginning. Nose-bleed or a little bleeding from the gums or a little blood in the stool may be noted. The skin bruises easily and slight blows and knocks which ordinarily would cause no trouble may leave an ecchymotic spot. The sleep is disturbed and the child becomes nervous and irritable. As the disease progresses the spleen becomes enlarged and the attention of the mother is usually called to the enlarged abdomen and the splenic tumor. In other cases the enlargement of the lymph nodes is noted.

**The Developed Stage.**—When the disease has reached its full stage of development, if such an expression may be allowed, the picture is a striking one. The pallor is extreme. The skin is flaccid, has lost its transparency, and is of a dirty, muddy appearance. There is a tendency to perspire. There may be occasionally a little icterus. There is a liability to eczema and sometimes there are pemphigoid eruptions. Purpura is common. The purpuric spots may be rather small and numerous or they may be larger and resemble bruises. Rarely there are lymphomatous nodules in the skin. These are small, whitish masses, varying in size from the size of a grain of wheat to a centimetre in diameter. When seen they are usually scattered all over the body.

As the disease progresses there is more or less edema and this is most noticeable about the face and extremities. Later on there may be effusions into the serous cavities.

The enlarged nodes which are usually present make a striking picture. There are generally several groups affected, in some cases practically all the lymphoid tissue of the body is involved. The cervical nodes are probably the most noticeable and the large collar of nodes may interfere with the movements of the head. The axillary and inguinal nodes are also easily felt. They are hard but not tender and are movable. The intrathoracic nodes may be enlarged and produce pressure symptoms similar to those mentioned in Hodgkin's disease. The mesenteric nodes may be enlarged and easily palpable. The tonsils and the lymphoid tissue about the fauces and mouth may be very much affected and be a noticeable feature of the disease.

The *spleen* is enlarged in all cases that have lasted any length of time. When it is large fissures may be felt along its margin. It may extend to the umbilicus or even farther. There may be tenderness over the spleen. The abdomen is distended and the child pot-bellied. The *liver* is often somewhat enlarged and may be very much so. The *urine* early in the disease shows no especial changes, but later there are albumin and casts. The heart is rapid and weak. There is dyspnea on the slightest exertion and there may be cough. The digestive symptoms are constant and generally pronounced, the loss of appetite and a tendency to diarrhea being most constant. There may be obstinate constipation; in some cases there may be hemorrhage of the bowel. The hemorrhagic tendency becomes more and more marked and may cause great weakness. The hemorrhages may occur from any mucous membrane or be subcutaneous.

There may be disturbance of vision due to leukemic retinitis, and there may be deafness. Nervous symptoms are usually noticeable. There is somnolence in some cases which may deepen into coma. There may be delirium. Pain may be complained of by older children, that in the spleen, the extremities, and head being most marked. The general weakness is extreme and there may be attacks of fainting. There may be fever ranging from  $101^{\circ}$  to  $103^{\circ}$  F., with even greater variations.

The clinical form of the disease and the blood findings are not marked as one would expect. But few cases remain pure to the end. Sooner or later they become mixed cases and show changes in nodules and bone-marrow as well. There may occasionally be noted cases of acute lymphatic leukemia where the lymph nodes are enormous, enlarged, while the spleen is comparatively small. In the chronic lymphatic cases the spleen is always enlarged, sometimes enormous so. The diagnosis lies in the blood examination, which should be made in all anemic cases.

**Prognosis.** Prognosis is always bad. Cases of recovery have been said to occur, but need not be looked for. Remissions may occur and the patient seem better for a time, but the disease returns.

**TREATMENT OF ANEMIA AND LEUKEMIA.**

The treatment of all forms of anemia and of leukemia may be considered together. In all cases the general management of the child is of great importance. Fresh air, sunshine, rest, and in mild grades of secondary anemia, carefully regulated exercises are required. In all severe anemias undue exertion should be avoided, and if there is marked shortness of breath or disturbance of the heart the child should be kept in bed. While in bed the child should, if possible, be given sun-baths or be in the fresh air. Porches, fire-escapes, and the like, may be utilized for this purpose. Excitement of all kinds should be avoided.

The *feeding* in young infants is important. The general rules for infant feeding may be followed where possible. In older children there should be five meals daily at regular intervals and the amount should be small enough to permit of perfect digestion. It is a good plan to give plenty of proteid food at breakfast—milk, eggs, or meat. If the diet in general contains too little proteid, somatose, eucasin, or some similar preparation may be added. In addition to meat and eggs, fresh fruit and vegetables may be used. Milk or mixtures of milk and cream should be given at the end of the meal to avoid spoiling the appetite, as may be done if it is given the first thing. In pernicious anemia Hunter advises but little proteid food and a great deal of carbohydrates and milk. Beef-juice, raw beef, glycerin extract of red bone-marrow may all be given to advantage, and the soluble beef preparations are of value in some cases, especially when combined with iron. Cod-liver oil is useful when it is well borne and does not disturb the appetite. A change of air and climate is very beneficial, especially a change to mountains or seashore for children who have a diminution of the number of red blood cells. All these children require sunlight.

In secondary anemia the cause should be sought for and removed. Intestinal parasites should be looked for, especially if there is eosinophilia. In pernicious anemia the ankylostoma or its eggs should be searched for in the stools. If suspected, thymol should be given.

The *drugs* of especial value are iron, arsenic, and tonics. In chlorosis and secondary anemia, iron is by far the most valuable. If the child can swallow a pill, freshly prepared Bland's pills are to be recommended. Aloes and nux vomica may be added to prevent constipation. Solutions of iron and manganese peptonate may be used and are particularly well borne. For young infants these are the best forms. The bitter wine of iron is also of value. Arsenic is valuable alone or to alternate with the iron.

In leukemia and pernicious anemia arsenic in the form of Fowler's solution should be given 0.06 c.c. (1 drop) three times a day, and gradually increased to three, five, or even more drops a day according to the age of the child. Care should be taken to avoid arsenic poisoning. A metallic taste in the mouth or puffiness about the eyes is an indication

to stop it. An arsenical neuritis may be the first symptom of arsenic poisoning. Iron is also used. Very recently several remarkable cases of temporary return to a normal blood state in leukemia following the use of Roentgen rays have been reported. The exposure was made daily over the spleen. Care should be taken not to burn the patient. The explanation would seem to be that the x-rays stop mitosis, as has been proven experimentally. The value of this treatment is not definite, but it looks promising. Recently Holding and Warren<sup>1</sup> have reported success with this method, especially in the splenic form of leukemia: 8 of 25 cases were said to be cured and 15 cases improved. In lymphatic leukemia the results were not so satisfactory, but improvement was noted. The x-rays seem also to have been beneficial in pseudoleukemia.

The pseudoleukemia of infants should be treated by iron or arsenic and iron alternating. Fowler's solution 0.06 c.c. (1 drop) three or four times a day is generally sufficient.

### PURPURA.

Purpura is the name applied to spontaneous subcutaneous hemorrhages. These may be either small, about the size of a pinhead (petechiae), or they may be larger and resemble a bruise (ecchymoses). When limited to the skin the condition is spoken of as *purpura simplex*. In severe cases, however, there are hemorrhages under the mucous membranes and into the internal organs and sometimes actual bleeding. These are called *purpura hemorrhagica*. These are only degrees of the same condition and the classification is unsatisfactory. Other ways of separating the purpuras are nearly as bad as the causes underlying the condition are but little understood.

**Etiology.**—Purpura may be regarded as a symptom sometime secondary to a known condition, but at other times apparently the chief symptom of some primary disease.

Symptomatic purpura occurs in the course of a large number of diseases and conditions the most important of which are as follows:

**Infections.**—It occurs in the course of many of the ordinary infectious diseases and infections; in smallpox, scarlet fever, measles, cerebrospinal fever, diphtheria; in hereditary syphilis, the septic infections, septicemia, pyemia, and malignant endocarditis. When it occurs in the infectious diseases the name "black" is prefixed by the laity as "black measles." The prognosis in these cases is very bad and the patients usually die. In making the prognosis it should be remembered that a cachectic purpura may appear late in the course of any of the above, especially measles, and that while this is apt to be in the severe and unfavorable case it is by no means always so.

**Cachectic.**—In this form the skin only is involved, as a rule. It is very common in infancy, particularly in institutions. When it occurs

<sup>1</sup> New York Medical Journal, November 11, 1905.



it has nearly always a very grave import. The commonest form is that which is seen in marantic infants over the abdomen, but sometimes on other parts of the body as well. It is also seen in bronchopneumonia, in empyema, ileocolitis, in tuberculosis, in nephritis, in Hodgkin's disease, when there are malignant growths, in the course of diseases of the blood, especially pernicious anemia and leukemia. Scurvy might be added to the list.

*Toxic.*—This is the form resulting from the administration of drugs, ptomaine poisoning, and in the course of jaundiced conditions. Among the drugs which may at times cause purpura are quinine, copaiba, mercury, belladonna, ergot, the iodides, potassium chlorate, antipyrin, arsenic, salicylic acid, and chloral.

*Mechanical.*—This is seen in epilepsy, whooping-cough, and very commonly after the removal of splints.

*Hemorrhage into the Adrenal.*—As Dudgeon has pointed out, purpura is a symptom of this lesion. (See Hemorrhage into the Adrenal, p. 849.)

*Neurotic.*—This is rare in young children; it may, however, be seen about puberty.

*Primary Purpura.*—This comes on without any apparent cause. The classification of cases is varied according to the author. Clinically the following forms may be considered: Purpura simplex, purpura hemorrhagica, purpura fulminans, Henoch's purpura, purpura rheumatica, and giant purpura without symptoms.

The condition needs further study on all points, as there are many discrepancies in the statements of observers. Some state that it is most frequent from two to ten years of age; others that it is more frequently seen from nine to fifteen years. In some collections of cases the sexes are given as about equal; in others boys preponderate.

*Pathology.*—This is obscure. The lesion consists in the hemorrhagic exudate in the skin, mucous membranes, and internal organs. The spleen may or may not be enlarged. Ulcers have occasionally been found in the stomach. The adrenals are generally the seat of enormous hemorrhages. There are no characteristic changes in the blood. There is usually an anemia of a secondary type, with or without leukocytosis.

Gangrene has occasionally been noted.

Various theories have been advanced to explain the condition. The principal ones are (a) that it is an infection, (b) that it is due to vasomotor changes, and (c) that it is due to endarteritis.

*Symptomatology.* **PURPURA SIMPLEX.**—In this form the hemorrhage is limited to the skin. The child may go to bed well and in the morning the petechiæ be noticed. More frequently there are prodromes consisting of general indisposition. After two or three days or even longer the purpura appears. At the same time there are liable to be disturbances of digestion, nausea, vomiting, and in some diarrhea. There is usually some fever, the temperature ranging from 100° to 103° F. The purpura consists of fine petechiæ and small ecchymoses. These generally appear first on the legs and then on the remainder of the body. At first

they are of a bright red or purplish color, but soon turn darker and become bluish black. They do not disappear on pressure. There may or may not be joint pains. The disease lasts from one to four weeks. Relapses are common. The outlook is good, nearly all the cases recovering. The prognosis should always be carefully given, as sometimes a mild case terminates rather suddenly in death.

**PURPURA HEMORRHAGICA.**—The name *morbus maculosus Werlhofii*, so often applied to this, really belongs to the disease which Werlhof described as *giant purpura without symptoms* (p. 832).

Purpura hemorrhagica is a severe disease. It may bear some resemblance to typhoid in its fever, course, prostration, and duration. The temperature ranges from 101° to 103° F. or more. The prostration is usually extreme. There are nausea, vomiting, and generally diarrhea. There may or may not be albuminuria. The hemorrhages are the striking feature of the disease. These may come on at the same time as the purpura or even precede it. The skin is mottled with petechiæ and ecchymoses. They vary from the size of a pinhead to half an inch in diameter. Their color varies from a red-wine color to a blackish red. They do not disappear on pressure. At times they may be painful or may itch. The purpura may be present on the mucous membranes. Slight external wounds cause a profuse hemorrhage like that described in hemophilia. Bleeding may take place spontaneously from any mucous membrane. Bleeding from the nose is the most common. It was present in 77 out of 100 cases (Barthez and Sannée). Bleeding from the mouth is common, especially from the gums. The hemorrhage may come from the tonsils or pharynx. In these cases the breath is very fetid. Hemorrhage directly from the stomach is more rare. Blood may be swallowed from bleeding in the mouth or nose and then vomited. Intestinal hemorrhages may also take place. Black stools result, but these may come from swallowed blood. If the blood passed from the bowel is bright red it is certainly from the lower part of the intestines. Hematuria may be present, but is not of very frequent occurrence. Hemoptysis is extremely rare in purpura. Bleeding may take place from the female genitalia. There may be retinal or choroidal hemorrhages. Intracranial hemorrhages are rarely seen. Edema may be present. Its location and extent vary. Pains may be complained of in almost any part of the body. Headache, backache, and pains in the abdomen are the most frequent. The anemia from the repeated hemorrhages may be extreme.

There may be marked nervous symptoms in some cases. These may be merely general nervousness and anxiety or in other cases there may be delirium, stupor, or even coma.

The *course* of the disease is variable. It lasts from one to six weeks. In some the patients after a few days pass into a typhoid state. They should not be confused with typhoid fever with a purpuric eruption. These cases are generally fatal. Purpura hemorrhagica is always a serious disease, but especially so in the weak, the very young, and when there are symptoms suggestive of septic infection.

*Diagnosis.*—The diagnosis is easy. Typhoid may, of course, be distinguished by means of the Widal reaction.

**PURPURA FULMINANS.**—This is a very acute fatal form of purpura rarely seen. It occurs most frequently under five years of age; older individuals may be affected. Many cases of "black measles," "black scarlet fever," and "black smallpox" dying rather suddenly without other eruption than the purpura seem to belong to this class. Its occurrence in unvaccinated infants suggested that it might be smallpox in some instances, but other cases of smallpox have not been noted after the purpurial eruption.

Large hemorrhages have been noted in the adrenals in some of these cases. The cases are usually sporadic, but a small epidemic has been reported. The child is taken suddenly ill with a chill or convulsion, vomiting, high temperature, and marked constitutional disturbance. The purpura comes on with extreme rapidity, covering the body in a few hours or a day. There may be vesicles filled with blood. The purpuric eruption may affect the mucous membranes, but actual hemorrhages are not common. There is delirium or stupor and coma. Albumin is found in the urine. The spleen is usually enlarged. Death may take place in ten or twelve hours, or the child may live two or three days. The patients do not live over five days.

**HENOCH'S PURPURA.**—This remarkable symptom-complex was first described by Henoch, and recently Osler has called attention to it and similar conditions in his articles on the visceral manifestations of the erythema group.<sup>1</sup> The condition is most frequently seen in childhood, has a tendency to recur at varying intervals, and may be seen in adult life. The symptoms may be grouped under three heads—skin, visceral, and arthritic. The most frequent *skin* lesion is a purpura, but there may be urticaria, circumscribed edema, or erythema exudativum. Any or all of these may be present or only one. They are liable to be most pronounced at the period when the visceral and joint lesions are most marked, but not necessarily so.

The *visceral* symptoms are numerous. Most important of these are gastroenteric crises consisting of colic. These attacks of pain may or may not be accompanied by vomiting, diarrhea, or the vomiting of blood; any one or all three may be present. These attacks last from a few hours to days.

Occasionally there may be cerebral symptoms. The patient may be slightly or markedly delirious. Hematuria and nephritis may occur, but are rare. They may apparently be causes of death. Hemorrhage from the mucous membranes occurs in some cases. Pulmonary symptoms, cough, bronchitis, and emphysema are occasionally present.

The *joint* lesions consist of swelling of the joint, of the synovial sheaths, or of the periarticular tissues. One or more joints may become affected. Sometimes there may be a severe polyarthritis like an acute rheumatism.

The attacks recur at intervals of weeks, months, or even years. The

<sup>1</sup> American Journal of the Medical Sciences, January, 1904.



colic and joint pain are usually present in most of the attacks. The most interesting point is that the skin manifestations vary; in one attack they may be purpura in a second urticaria and so forth. The prognosis is none too good. Over 25 per cent. of Osler's cases died.

**PURPURA RHEUMATICA (Schönlein's Disease).**—This is not as common in children as in young adults, but it does occur, rarely under five years, however. These cases have been regarded by some as rheumatism plus purpura, by others as a separate disease. The clinical picture is so distinct as to be easily recognized. It is characterized by a multiple arthritis resembling rheumatism or by actual rheumatism, and in addition there is purpuric eruption consisting of petechiae and small ecchymoses; there are urticaria and skin lesions which might be put down as erythema exudativum or erythema multiforme. In some cases there may be edema and this may at times be very marked. The location and amount of edema are variable. There is frequently fever. This is not very high, but generally ranges from  $101^{\circ}$  to  $103^{\circ}$  F. The disease frequently begins with a sore throat. There may be albumin in the urine. The disease lasts about three weeks and the tendency is nearly always to recovery. Relapses are common.

The diagnosis is easy. The joint symptoms, the urticaria, the erythema with the purpura form a clear picture.

**GIANT PURPURA WITHOUT SYMPTOMS.**—This rare form of purpura was described by Werlhof in 1735 under the name of Morbus Maculosus Hemorrhagicus. By singular misfortune the name morbus maculosus Werlhofi is usually applied to the ordinary severe form of purpura hemorrhagica. There is unfortunately a great difference of opinion in regard to all forms of purpura and there are wide variations in the application of terms. The disease in question is most frequently seen between the ages of five and fifteen, although it may be seen either earlier or later. Its distinguishing characteristics are that its onset is sudden with a purpuric rash and sometimes with hemorrhages from the mucous membranes of the nose, stomach, etc. There is neither fever nor joint trouble, and no symptom but the bleeding. The purpura consists of petechiae, and, what is most important, of very large ecchymoses; these may be several inches in diameter. They last from one to two weeks and disappear. Occasionally they may last longer. The purpura may recur. Practically the outlook is always good. Hemorrhage occurring in an internal organ, however, may cause death.

The diagnosis is easy; the size of the spots and absence of fever and symptoms are the principal points to be considered. From trauma is at times difficult and may depend upon the history, which may be important from a medico-legal standpoint. The finding of hemorrhagic spots on the mucous membrane will help as showing the disease. Such cases have been regarded as mild atypical scurvy, but they occur at an age when scurvy is rare and there are none of the other symptoms.

**Treatment.**—The treatment of secondary purpura consists in the management of the original disease and, if practicable, the suggestion made for primary purpura may be added.



In *primary* purpura, especially in the severer forms, the child should be kept quiet in bed and guarded from all injuries and bruising. The diet is perhaps the most important thing. This should be on the same lines as that recommended in scurvy. Fresh fruit juices, fresh vegetables, fresh milk and meats may be used as freely as possible. In the severer cases orange-juice may be added to the milk or other light diet that is being used. The diet should be kept up during convalescence. Some cases seem to be greatly benefited by it, while others are but little affected. A great many drugs have been tried. Adrenalin may be given in doses of 0.06 c.c. (1 drop) or more of the 1:1000 solution several times a day. The mineral and vegetable acids and the astringent drugs, such as gallic acid and hamamelis, are recommended. The very severe forms should be treated symptomatically. During convalescence careful feeding and tonics should be used. Iron, if the child is anemic, is one of the most important.

#### HEMOPHILIA.

Hemophilia is a curious, rare disease of a family and hereditary nature, characterized by a tendency to grave hemorrhage from very slight causes. Popularly these patients are called "bleeders."

**Etiology.**—The disease runs in certain families and has been known to persist through seven generations, covering a period of two hundred years. Isolated cases have been reported, however, where there was apparently no family taint. It has been looked upon as a stigma of degeneration. It occurs more frequently in boys than in girls. Dunn<sup>1</sup> has collected 780 cases, 717 being in males and 63 in females. "The female members of bleeder families are *par excellence* conductors of the disposition. The daughters in bleeder families are comparatively exempt from the tendency, while the sons are liable to it. They may themselves be healthy and marry healthy husbands, yet the bleeder habit is likely to be conducted to their sons. The daughter of a bleeder family, herself a bleeder, is not more likely to transmit the tendency than her non-bleeder sister. A son of a bleeder family, himself a bleeder, should he live to beget children, does not often conduct the disease to his children, but to his grandsons through his daughters. Again, should he have non-bleeder brothers, their grandsons seldom bleed." (Dunn.) The families are exceedingly prolific and a little over half of the children have the disease. It is more common in cold climates than in warm and seems to be unknown in the tropics. It is found in certain communities with great frequency, supposedly from intermarriage of members of hemophilic families. It is said to be most frequent in Germans and Hebrews. It usually begins in the first two years of life, and is rarely seen to begin after ten years of age and practically never after twenty. Grandilier gives 65 cases in boys. Of these 62 began before the tenth year and 40 in the first year. Joint affections and

<sup>1</sup> American Journal of the Medical Sciences, 1883, vol. lxxxv.

asthma may be met with in these families. Similar transmission of disease through the daughters has sometimes been seen in cases of diabetes insipidus, Duchenne's paralysis, color blindness, great thirst, etc.

**Pathology.**—This is unknown. It has been supposed that there is thinness of the coats of the arteries and degenerations of the walls. This, however, does not stand on a very firm basis. There is no change in the blood except that the coagulability is delayed. After the hemorrhages there may be a temporary secondary anemia.

**Symptomatology.**—The symptoms are very simple. Following slight injuries, as abrasions, scratches, erosions, superficial cuts, and the like, there is severe and sometimes uncontrollable hemorrhage. The bleeding is more of an oozing than of violent hemorrhage, but the quantity of blood lost in a short time may be enormous. Cases have been reported where the amount was a pint or even a quart in a few hours. The bleeding may last a week, with remissions and intermissions. There is a tendency to bleed from mucous membranes. Hemorrhage from the nose or from the bowel may take place. There are apt to be petechiae, ecchymoses, and hematomas. They may result from trifling bruises. When the hemorrhage is not traumatic prodromes are sometimes observed. There is a rush of blood to the head, acuteness of hearing or of sight, buzzing in the ears, deafness, disturbances of vision, epileptiform convulsions, or attacks of laughing or excitement. These pass off when the hemorrhage begins. It is interesting to note there are, as a rule, no disturbances of menstruation beyond a tendency to early and rather profuse flow, nor is there any unusual bleeding at childbirth.

There may be effusions of blood into the joints, the order of frequency being the knee, foot, hip, shoulder, and elbow. The affections of the joint include acute effusions with or without fever, arthropathies with swelling and deformity which may be mistaken for other joint troubles, and extensive joint changes, often with ankylosis, which resemble a form of arthritis deformans.

The symptoms following the bleeding are those of any severe hemorrhage. Death may take place with convulsions. In favorable cases the patient is liable to fall into a deep, prolonged sleep from exhaustion.

Three forms of the disease have been described: 1. The severe form, in which there is a tendency to severe, spontaneous or traumatic hemorrhages, associated with swelling of the joints. This is seldom seen in females, generally lasts through life, and usually is the cause of death. 2. The intermediate, in which there is no tendency to joint affection or traumatic hemorrhages, but frequent spontaneous ones from mucous surfaces and subcutaneous ecchymoses. This form frequently appears at puberty. 3. A mild form seen only in females; there are ecchymoses and early and prolonged menstruation.

**Diagnosis.**—This is made from the bleeding, which is spontaneous or follows slight causes, the difficulty of stopping such hemorrhages, and the history of the disease in the family and of previous attacks. The history of the presence of the joint troubles may be of some value. Care should be taken to exclude the hemorrhagic diseases of the new-

born, which are of a different nature. Bleeding from the umbilicus is rarely hemophilia. Scurvy may be mistaken for hemophilia. The treatment by dietetic means soon clears up the doubt. Leukemia or severe anemias can be excluded by a blood examination. Purpuric conditions are acute, and if the child lives there is no tendency to hemorrhage left behind.

**Prognosis.**—Prognosis is worse in boys than in girls. The longer a bleeder lives the less liable is he to die of his peculiar disease.

Grandidier gives the following interesting table of 212 fatal cases—197 males, 15 females:

	Males.	Females.	Total.
Within the first year . . . . .	22	7	29
From one to seven years . . . . .	89	3	92
“ eight to fourteen years . . . . .	39	1	40
“ fifteen to twenty-one years . . . . .	24	3	27
“ twenty-two to twenty-eight years . . . . .	8	..	8
“ twenty-nine to thirty-five years . . . . .	6	1	7
“ thirty-five to forty-five years . . . . .	8	..	8
Over fifty years . . . . .	6	..	6

Almost all of the cases observed die before they are ten years of age, nearly all the remainder before they are twenty, while if they go past that age they are apt to die of some other affection.

**Treatment.**—Prophylaxis consists in preventing the marriage of bleeders where possible, especially of the daughters. After the child is born it should be guarded from injuries of all kinds. As the disease is not seen in hot climates the removal to some tropical place has been advised and has been successful in a few cases.

When hemorrhage occurs the child should be kept at absolute rest. If the part is accessible pressure should be applied. All sorts of styptics have been advised and may do good. Tannic acid and perchloride of iron have perhaps given the best results. Of course, operative measures are to be advised against.

Adrenalin 1: 1000 may be applied directly to the spot or given internally in hemorrhage from the stomach. Cocaine solutions may also be used in place of this. Ergot has been used with success in some cases. Sulphate of soda in small doses, 0.13 gm. (2 gr.), repeated every two hours has been recommended. The liquor of perchloride of iron in 2 c.c. (half-drachm) doses has been used by Legg. Gelatin in 5 per cent. solutions injected subcutaneously has been recommended. Care should be taken to have it sterile. Gelatin solutions by mouth may be tried. A salt solution may be tried by enema, but seems to be of little or no definite value.

## CHAPTER XXXIII.

### THE THYMUS—STATUS LYMPHATICUS—ADENITIS—HODGKIN'S DISEASE—THE SPLEEN.

#### THE THYMUS GLAND.

THE gland extends from the notch of the sternum or somewhat above it as far down as the second, third, or fourth costal cartilage. Its width varies from 1 to 2.5 cm. (half an inch to about an inch). It varies considerably in size in different individuals, according to their age, size, and state of nutrition. It increases from birth to about two years rather rapidly, slowly from that time until puberty, when it remains stationary until twenty-five or thirty is reached, then it atrophies and is replaced by fat and connective tissue. It weighs about 3 gm. at birth, about 5 gm. at the second year, and from 7 to 12 gm. later on, according to some authorities, while others give 14 gm. at birth, 20 gm. at the ninth month, and 25 to 30 gm. at the second year. The latter figures are perhaps the more reliable.

The function of the thymus is not definitely known. Briefly stated the chief theories are as follows: Kölliker and Beard think that it is the parent source of the leukocytes; Chiari and Ziegler that it acts in place of the lymphatic system in intrauterine life until it is replaced by other lymph-forming organs (pharyngeal and faucial tonsils) taking up its function. It is intimately connected with the lymphatic system, as it is enlarged in general lymphatic enlargement. It atrophies in the atrophy of the lymphatics such as that following thyroidectomy. It has some relation with the spleen. Friedleben found that as the spleen got larger the thymus grew smaller. When there are numerous nucleated red blood cells in the spleen there are few in the thymus and *vice versa*.

It seems to bear a close relation to the state of nutrition, the development and growth of the individual. Both macroscopically and microscopically it is a good index to the state of nutrition of infants. In well-nourished infants the thymus is well developed, in moderate atrophy it is small. The most marked pathological change in it is found in the extreme atrophies of infancy, both primary (marasmus) and that secondary to wasting diseases. In these cases it is atrophied and much of the gland replaced by fibrous tissue.<sup>1</sup>

The thymus may be altered by changes in general diseases, such as syphilis and tuberculosis, and may be the seat of tumors of various kinds and of abscesses. Hemorrhages are of frequent occurrence, especially in infants who have been asphyxiated. It has been found to

<sup>1</sup> Stokes, Rothrb, and Robner, American Journal of the Medical Sciences, November, 1892.



be hypertrophied in some, but not all, cases of acromegaly (43 per cent.), gigantism, Graves' disease, chlorosis, leukemia, Hodgkin's disease, epilepsy, and somewhat in infections, although authors differ on this point. It is also hypertrophied in thymic asthma and in Paltauf's status thymicus (status lymphaticus).

It is atrophied in atrophic conditions of the body and in rickets. In idiots, Bourneville found it was present in only 27 per cent. Katz found it present in every case in sixty-one autopsies on mentally sound children.

**Hypertrophy of the Thymus. Sudden Death.**—The thymus may be enlarged in the course of various diseases as mentioned above, or it may become enlarged alone. When this happens and the gland reaches a sufficient size it causes symptoms and may be a cause of sudden death in young children and infants, or in older individuals with Paltauf's status thymicus. The history of these cases is usually that the infant has been put to bed perfectly well or sometimes with a slight cyanosis. When next seen it is dead with marked lividity of the body. Autopsy reveals a large thymus weighing an ounce or an ounce and a half (30 to 45 gm.). There are apt to be hemorrhages in the gland. It is important to bear this form of sudden death in mind from a medicolegal point of view.

**Thymic Asthma.**—The enlargement may be slight and may come on gradually and the condition may last months. Some of the patients with enlarged thymus glands die suddenly after having had symptoms for some time. The symptoms are those of intrathoracic pressure. There is a pallor of the face with usually a slight edema, especially marked in the parotid region, under the jaw, and about the eyes. The conjunctivæ are suffused and may be infiltrated with blood. The lips are cyanosed to a greater or less extent, as are also the finger-nails. The respiration is labored and noisy with inspiratory stridor. In some this seems to be the result of direct pressure upon the trachea and in others from spasm of the larynx.

There is dulness over the upper part of the sternum and the gland may, in some cases, be felt above it. The head should be extended in making percussion.

**Diagnosis.**—The diagnosis of the exact condition is difficult from tumors or enlarged bronchial lymph nodes. This, however, is of no great practical importance. If the dulness is very irregular it is usually due to enlarged lymph nodes.

**Treatment.**—The treatment is to remove the offending mass if symptoms are sufficient to cause manifest trouble. This has been done successfully a number of times with perfect recovery and with relief of symptoms.

#### STATUS LYMPHATICUS.

Under this name or that of Status Thymicus a condition of considerable interest has been recently much discussed. Paltauf called attention to

although pulled out immediately; shower baths, and use of chloroform.

**Etiology.**—This condition clinically may be seen at especially in young children. At puberty there seems to be for the lymphoid tissues to undergo atrophy and in certain instances the individuals outgrow hypertrophy of lymphoid tissue. As noted above, however, it may persist and be followed by death.

In some cases the condition seems to be present at birth until about puberty if the child lives. In others it seems to develop later, enlargement of the lymphatic structures appearing from slight causes and remaining. These acquired cases are common in the poor in institutions and tenements, and are associated with rickets. They should be distinguished from scrofulous or strumous children where the lesion is tubercular.

**Pathology.**—The status lymphaticus consists in enlargement of the lymph nodes and of all the lymphatic structures, of the thymus gland, with also an increase in the lymphoid tissue of the bone-marrow. These changes are frequently seen in children with rickets. There may also be a hypoplasia of the blood vessels, especially the aorta.

**Symptomatology.**—The children are usually pale, and poorly nourished, but often the flesh seems more or less flabby. The cervical and faucial tonsils are hypertrophied. The ring of lymphoid tissue about the fauces is prominent. The lymphoid follicles on the tongue and about the tongue are enlarged. The circumvallate papillae on the tongue are prominent. The lymph nodes over the neck are swollen and palpable. The thymus is increased in size and the upper part of the sternum is easily made out and is enlarged and easily palpable. The thyroid is said to be enlarged in some cases, but this has not been present in the cases where

bring on fatal convulsions, and children who die after slight indiscretions of diet are of this type.

**Treatment.**—This is not very satisfactory, but, fortunately, in most instances the status lymphaticus is outgrown. Good hygiene and good food are important, and plenty of fresh air and sunshine necessary. Enlarged tonsils and adenoids should be removed. It should always be borne in mind that these children do not take anesthetics well and that chloroform is especially dangerous.

Cod-liver oil in cold weather and the syrup of the iodide of iron give the best results in the way of drugs. Iodide of potassium may be given a trial.

### SIMPLE ACUTE ADENITIS.

This is an acute inflammation of the lymph nodes. The lesion is secondary to inflammation or irritation elsewhere in some adjacent tissue which is drained by the chain of lymph nodes that is affected or is part of some general infective process. The external and internal nodes are both affected. The external suppurate frequently, but the internal apparently quite rarely. The bronchial lymph nodes are affected in lesions of the lungs and bronchi (see p. 351); the mesenteric in intestinal disorders, etc.; but while these enlarged nodes are found at autopsy they are not large enough, as a rule, to be made out during life and do not play any very marked role in ordinary practice, except as due to tuberculosis, Hodgkin's disease, or lymphosarcoma, which may cause pronounced symptoms.

The external nodes are frequently enlarged. Roughly speaking, about three-fourths of the cases are seen under two years of age. Being near the surface, they can readily be palpated. The cervical nodes are the ones most often affected, the axillary and the inguinal more rarely.

In the infectious diseases the superficial nodes are quite regularly enlarged, usually from the result of the local inflammations. In rubella, however, the posterior cervical nodes are enormously enlarged and are of some diagnostic importance. The commonest causes of adenitis are catarrhal conditions of the nose, throat, and mouth. The primary cause may be so slight as to be easily overlooked. Carious teeth and stomatitis, especially ulcerative stomatitis, are frequent causes of the submaxillary nodes being inflamed. Eczema of the scalp and the irritation due to lice as well as other diseases of the scalp are frequent causes of the posterior cervical nodes being affected. Otitis and injuries should not be forgotten. The axillary lymph nodes are enlarged from vaccination and the inguinal from vaginitis.

**Pathology.**—The lesions consist in a swelling of the node due to acute congestion and to a hyperplasia of the lymphoid cells. The nodes feel hard, and, on section early in the disease, are homogeneous in their appearance. The microscope shows a simple hyperplasia. They may remain hard and firm for indefinite periods, especially if there have been recurrent attacks or continuous irritation. If the cause is quickly

removed the nodes usually subside after a few weeks if they do not suppurate. When suppuration occurs the nodes soften, the surrounding tissue becomes infiltrated, and a localized cellulitis results. The process is usually unilateral or, if it involves both sides, one side is almost always much worse than the other. If suppuration occurs it is liable to last on one side and often only a single node may break down.

**Symptomatology.**—The symptoms include the disease which is the cause of the trouble. Frequently there is diphtheria, scarlet fever, or some other infection. There may be a slight pharyngitis which would pass unnoticed were it not for the extreme nodular enlargement which may follow. In these cases the swelling is frequently at the angle of the jaw. The lymph nodes often enlarge very rapidly, but the swelling may come on rather gradually. They are painful and tender and there may be redness of the skin. Suppuration when it takes place usually starts during the first or second week, but it may be delayed for three or even four weeks. After that time suppuration seldom occurs. When it does occur there is decided redness of the skin and the swelling becomes more diffuse. After a few days the little abscess which forms points and if not opened breaks through the skin. After the pus is discharged the healing is usually quite rapid. When suppuration does not occur the nodes remain swollen from a week to two months, gradually becoming smaller and harder. They may disappear entirely or a little hard node may be left. These nodes are liable to enlarge later on from a recurrence of the primary trouble. When there have been several recurrences, or where the irritation is kept up for a long time, as that from a neglected carious tooth, the node may remain hard throughout life as an evidence of old inflammation.

At the height of the disease there is usually fever.

**Diagnosis.**—Diagnosis is easy. The occurrence of enlarged lymph nodes in a child under two years of age or in older children where there is a definite cause renders error unlikely. After two years of age tuberculosis of the nodes is common. This is a much more chronic process. The location of mumps in the parotid region with the lobe of the ear as the centre of the swelling and the history of exposure are usually sufficient to differentiate this disease. The other node affections are chronic.

**Treatment.**—Where the local cause is apparent it should be treated. The nose, throat, or teeth should receive attention. Catarrhal conditions of the mucous membranes should always receive prompt treatment.

For the nodes themselves local applications of heat or cold may be applied: cold, if there is swelling and congestion; heat, if the process is one of pus formation. Applications of ichthyol, 5 to 10 per cent., either as an ointment or with glycerin, may be used and often give considerable comfort. If suppuration takes place the resulting abscess should be opened under the usual aseptic precautions. It is best to wait until the abscess "points" and then make an incision. When suppuration does not take place or to hasten absorption in the remaining enlarged nodes, iodide of potassium has been advised. It may be given to



infants in doses of 0.06 to 0.19 gm. (1 to 3 gr.), diluted in water or milk, four times a day.

The usual painting with tincture of iodine is valueless.

### SIMPLE CHRONIC ADENITIS.

This is not common in severe forms, but mild grades of chronic adenitis are frequently seen. It results usually from recurring attacks of acute adenitis or from chronic inflammatory conditions of the mucous membranes with which the nodes are connected. Skin lesions or long-standing suppurations may also cause it. The posterior cervical nodes are often found enlarged in poor children with chronic scalp disease. In children with the so-called status lymphaticus it is one of the features of the condition which has been described. The tonsils are frequently enlarged and adenoids may be present.

**Symptomatology.**—The manifestations of the disease are simply slight swelling of the lymph nodes, the neck being the most usual site. The nodes enlarge and remain so for a few months and then generally subside. They may remain for years. They are, as a rule, not tender. They do not tend to suppurate.

**Diagnosis.**—This is chiefly from tuberculous nodes or from Hodgkin's disease. The age and the very slow course are the principal features. Most of the cases where the enlargement is sufficient to cause doubt are in infants under three. The removal of a node for diagnostic purposes is permissible if there is strong suspicion either of tuberculosis or of Hodgkin's disease.

**Treatment.**—Treatment consists in removing the cause where it is apparent. Enlarged tonsils and adenoids if present should be removed and any catarrhal conditions which may exist should be treated. A change of climate may be desirable.

Internally, cod-liver oil may be given in cold weather. Iron in the form of the syrup of the iodide, or iodide of potassium, or arsenic in the form of Fowler's solution may be used.

**Tuberculosis of the External Lymph Nodes.**—This is treated of in detail under the heading of Tuberculosis (p. 351) and does not need further elaboration here.

**Syphilitic Adenitis.**—Syphilis, especially late hereditary syphilis, may occasionally be a cause of marked swelling of the lymph nodes. The enlargement is generally universal, but may be localized. In some instances it may be associated with lesions in the adjacent tissues. The recognition that it is syphilitic rests on the finding of other manifestations of that disease and on its rapid improvement on antisyphilitic treatment. All these points are discussed in full in the chapter describing the disease and its treatment (see p. 563).

## HODGKIN'S DISEASE.

This is known under a great number of names and is confused with other conditions. Among the most frequent synonyms are *Adenar* (Trousseau); *Anemia Lymphatica* (Wilks); *Pseudoleukemia* (Coblenheim), and *Generalized Lymphadenoma*.

It is a disease characterized by a progressive enlargement of the lymph nodes and the spleen and the formation of nodules in the internal organs (liver, spleen, kidney, etc.), and sooner or later a secondary anemia and cachexia.

It is a disease of early life and the majority of the cases occur in childhood. In Hodgkin's original report, in 1832, some of the cases reported were in children. In 43 cases collected by Clement Clarke 10 were under ten years of age. It is more frequent in boys than in girls.

**Etiology.**—The exact cause is unknown. It has been suggested that it is the result of an acute infection of some unknown agent, but this has not been proven. Some recent writers have thought that it was due to the tubercle bacillus, but while secondary infections with this organism are common the original changes in the nodes can still be made out histologically. In some cases there is no tuberculous complication.

**Pathology.**—The morbid anatomy consists in enlargement of the lymph nodes, both deep and superficial, and of the spleen as well in most cases. The nodes do not tend to break down unless there is secondary infection and there is no tendency to invade the surrounding tissue as in lymphosarcoma. There are lymphomatous nodules in the organs and there is involvement of the marrow of the long bones. According to the studies of Dorothy M. Reed<sup>1</sup> the histological changes are as follows: In addition to the proliferation of the endothelial and reticular cells and the formation of lymphoid cells there are seen characteristic giant cells which differ from the giant cells of tuberculosis. There is proliferation of the connective-tissue stroma which gives rise to the hardness of the nodes noted as the disease progresses. There are also numerous eosinophiles found in the nodes. It should not be confused with sarcoma of the lymph nodes which has a different histological structure, nor with tuberculosis of the lymph nodes (Fig. 17-2).

There do not seem to be any special predisposing diseases. Tuberculosis is not found in the family history any more than is usual. The patient is usually in good health, but there may be chronic tonsillitis or inflammations of the eye or ear before the disease manifests itself.

**Symptomatology.**—The disease starts almost always in the neck. The nodes become enlarged. They are first somewhat soft, but later become hard and firm. As a rule, they are not painful. The disease extends until the other superficial and the internal lymph nodes have become involved. The progress of the disease is steady, but there may be temporary remissions. The nodes do not tend to break down unless there is

<sup>1</sup> Johns Hopkins Hospital Reports, 1902, vol. x.

a secondary infection, and there is no tendency to involve the skin unless this happens. The disease may last months with the patient's general health good. Sooner or later, however, there is a marked secondary anemia with cachexia and pronounced weakness. This may come on in a few months or it may be delayed for years. There is irregular fever. This may be absent or may be continuous or may be occasionally of a remittent type. In three-fourths of the cases the spleen is enlarged. Other symptoms which may be present are pressure symptoms from the masses pressing on trachea, bronchi, nerves, ureters, etc. There may be bronzing of the skin.

**Diagnosis.**—The clinical picture is much the same in lymphosarcoma, but there is a greater tendency to involvement of adjacent tissues, and there is also a greater liability to pressure symptoms. The removal of a node under cocaine anesthesia for histological study is the most certain means of diagnosis.

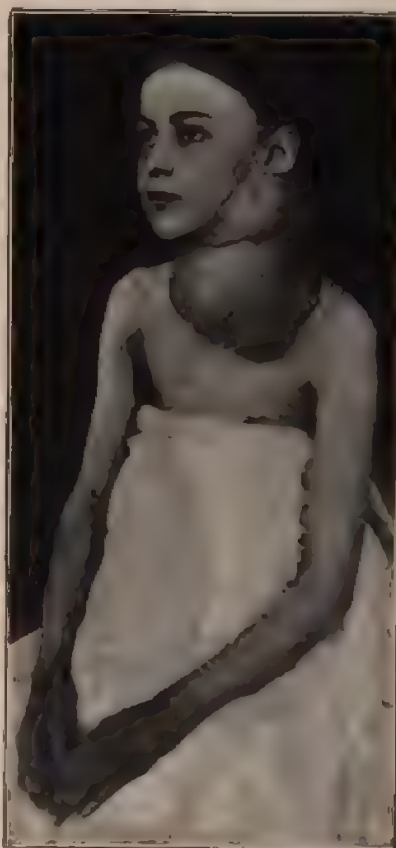
**Tuberculosis.** In early cases of tuberculosis this may be very difficult. If there is no fever tuberculin may be used. It is certain and harmless. There is usually tuberculosis in the lungs or elsewhere and there may be suppuration of the node or the matting together of the nodes. The removal of a node will clear up doubtful cases.

**Leukemia.**—Diagnosis in leukemia is easy, as a rule, from a blood examination, but there are rare cases where in a leukemia the leukocytes have fallen to normal or near it. The structure of the nodes is different.

**Prognosis.** This is bad. Sooner or later the cases become cachectic and die. The average duration of life after the appearance of the disease is from one to four years. Death is usually caused by a secondary tuberculosis or by progressive weakness with general anasarca.

**Treatment.**—Treatment is not very satisfactory. If the case is diagnosed early and the enlarged lymph nodes are only on one side of

FIG. 172



Hodgkin's disease. (Case of Drs. Sherman and Gaylord, Archives of Pediatrics.)



the neck, removal of the nodes is thought to have some influence on prolonging life and preventing, at least for a time, the progress of the disease. Osler thinks that the Roentgen rays may have some influence in selected cases. The patient should lead a regular hygienic life, with plenty of fresh air and good food. Of the drugs used arsenic is the favorite and some results seem to have been attained by it. It may be given in fairly large doses over rather long periods without causing any trouble. In some cases it produces pigmentation of the skin and in others neuritis may result. Fowler's solution may be given in from 0.13 c.c. to 0.3 c.c. (2 to 5  $\text{m}$ ) doses three or four times a day. The syrup of the iodide of iron may be tried if the arsenic disagrees, or other tonics may be given, such as cod-liver oil and quinine. Phosphorus has been recommended. It is given in doses from 0.000325 to 0.00065 gm. ( $\frac{1}{2000}$  to  $\frac{1}{1000}$  gr.). The effects should be closely watched.

### DISEASES OF THE SPLEEN.

The spleen is of somewhat more value in diagnosis in infants and children than in adults. In the young it is more readily affected and as a general rule, more easy to make out. The normal position of the spleen should be borne in mind. It lies with the upper border reaching about the ninth rib and the lower border about the eleventh rib. Toward the back it extends as far as the posterior axillary line or a little back of the end of the eleventh rib. In front it extends to the midaxillary line or a little farther, but normally does not pass a line drawn from the nipple to the end of the eleventh rib.

The splenic dulness if made out normally corresponds to the above. In infants the normal splenic dulness is so small that it may not be possible to make it out in all cases. At best it is uncertain, as it may be obliterated by the abdominal tympany or a large spleen made to seem smaller, while intestinal contents may give rise to dulness which may be mistaken for the spleen. An exudate in Traube's semilunar space may cause a fusion of liver and splenic dulness, and this may also occur in great enlargement of either or both organs.

Palpation is much more certain than percussion. With an infant it is best to have the child lying on its back on its mother's or the nurse's lap. If the spleen cannot be felt in this position the infant may be turned on the right side. The hand should always be warmed and then placed gently on the abdomen, and as soon as the muscles become accustomed to its presence an effort may be made to feel the spleen. The finger or one finger should be approached to the edge of the ribs between the middle and posterior axillary lines. In young infants and in those with soft abdominal walls the spleen may often be felt just under the edge of the ribs. The finger should be held in one place during several inspirations, when the spleen may be felt touching the finger during inspiration and disappearing during expiration. Then moderately rapid palpation



movements should be gently made. If the spleen is felt by this method the edge will be felt as it slips past the finger. If the spleen extends below the margins of the ribs it may be looked upon as enlarged unless pushed down by a pleural effusion. A moderately enlarged spleen extends 2 or 3 cm. below the margin of the ribs, but it may be so large as to extend to the brim of the pelvis and past the umbilicus. All grades may be seen. An acute enlargement rarely exceeds  $2\frac{1}{2}$  or 5 cm. (1 to 2 inches) below the edge of the ribs, and if a spleen is found larger it is quite safe to assume that it is a chronic enlargement. The inner border is generally thin and sharp. About the middle of it there is a notch.

The upper border of the spleen can never be felt except in the case of floating spleen.

It should be remembered that the spleen, if it moves during breathing, does so not directly downward, but diagonally toward the right pelvic brim. The spleen enlarges in this direction as well. This may be useful in differentiating tumors of other organs. Usually a tumor mass of other organs moves directly downward during inspiration. If not too large or fixed by adhesions the spleen may be moved laterally.

The liver is frequently enlarged at the same time and the two may lie so close together that it is impossible to make out the one from the other. Sometimes in marked enlargement of the liver the fissure made in the liver by the round ligament may be taken for the dividing line between the liver and spleen. This fissure usually is in line with the umbilicus.

The spleen may often be seen if enlarged. In a good light, with the abdominal wall held on a stretch by the hand of the physician, the spleen may be plainly seen to move with the inspirations. There may also be enlarged superficial veins and a slight violet color may sometimes be noted.

Over a very large spleen there may be heard a *bruit de souffle* or blowing murmur similar to that heard over a pregnant uterus. This may be elicited on pressure over the larger vessels. In most conditions where there is a large spleen the child has a peculiar pallid, brownish-yellow color. The skin has lost its transparency and become "muddy."

The position of an older child for examination should be the same as for an adult where it is possible to control the child. The child should be flat on the back with the knees drawn up or, where possible, what is much better, the upper part of the body should be sharply inclined against the back of the bed and a pillow. This latter position allows the legs to be kept down and out of the way, and at the same time permits of relaxation of the abdominal muscles, and has also the advantage of allowing the spleen to descend should it happen to be movable. When the spleen cannot be felt in this position, the child should lie on the right side with the legs flexed and the left arm over the head. The breathing is an important part and is best taught by imitation. The physician should show the child just how to take long, deep inspirations followed by com-

plete expirations. This may be made into a sort of play, and the child may often have his attention diverted from the examination by trying to keep time with the physician's breathing and similar tactics.

**Enlargement of the Spleen.**—Enlargement of the spleen may be either acute or chronic. As a general rule, it may be stated that all of the acute infectious fevers are attended with some enlargement of the spleen. This is generally only a hyperemia. In typhoid and malaria it is a constant and important diagnostic feature. In cerebrospinal fever it is only occasionally seen, and in mumps it is seldom enlarged. The spleen is enlarged in most of the chronic conditions met with in early life.

In *rickets* it is quite a constant feature in the active stages of the disease. In diseases associated with blood changes, as in leukemia, splenic anemia, and in the pseudoleukemia of infants, the spleen is very much enlarged. It is enlarged in Hodgkin's disease. In syphilis, in the early and more active stages, it is easily felt in the majority of the cases. It is also enlarged in some of the later cases. There may be circumscribed gummatous enlargements, which are seen late and are rare; or there may be merely a diffuse swelling of the organ, which is commonly seen.

In *acute miliary tuberculosis* the spleen is sooner or later enlarged. In the other forms of tuberculosis the spleen may or may not be affected. It may often not be enlarged at all. In other cases it may be the seat of extensive tuberculous deposits and be considerably increased in size.

The *amyloid spleen* is large, hard, smooth, and thick. It is met with in cases where there has been long-standing suppuration, especially in caries or necrosis of the bones. It may also be seen in chronic tuberculosis of the lungs and in syphilis.

The spleen may be the first organ to show amyloid changes. The diagnosis is made from the pre-existing condition of the patient. There is the history of the chronic disease, general cachexia, pallor of the skin, emaciation, and usually diarrhea, albuminuria, multiple hemorrhages, petechia and the like. In the early stage the spleen alone may be enlarged. If the condition has been existing some time the liver is also enlarged. This is the condition called "sago spleen" by the old writers.

#### CHRONIC PASSIVE CONGESTION OF THE SPLEEN.

Chronic Passive Congestion of the spleen occurs where there are disturbances of the portal or of the splenic circulation. This is met with in cirrhosis and more rarely in syphilitic changes in the liver, from monolocular or multilocular echinococcus cysts, and from hyperemia of the liver. It may occur from emphysema, cirrhotic conditions, or advanced tuberculous lesions in the lungs, and from acquired or congenital heart lesions. When the spleen is enlarged from chronic passive

congestion the liver is too, except in those rare conditions where there is disturbance of the splenic circulation alone.

### **SPLENITIS AND PERISPLENITIS.**

Inflammation of the spleen may occur from extension of a neighboring inflammatory process. The diagnosis is uncertain, but an enlarged spleen, pain in the splenic region, and the pre-existing inflammation are the parts to be considered. Perisplenitis may result from peritonitis, trauma, hemorrhagic infarcts, syphilis, or tuberculosis. The spleen is generally enlarged. The diagnosis is made by feeling the friction rub. It is less certain when the friction sound is heard, as it may be confused with pleurisy. If it is heard louder below than above, and especially if it is heard better with the stethoscope at the edge of the ribs than over the chest wall, one may think of perisplenitis. In chronic perisplenitis there may be adhesions and the spleen is no longer movable. There is more frequently a chronic thickening without adhesions.

*Floating spleen* may occasionally be met with as a congenital condition. The diagnosis is usually easy if abdomen is sufficiently relaxed to admit of satisfactory palpation. There is tympany over ninth to eleventh ribs. The spleen is felt elsewhere, generally under the left hypochondrium, but it may be as low as the pelvis. The spleen may be recognized by its shape, and if there are no adhesions may be pushed back in place. Care must be taken to differentiate fecal masses in the colon and tumors of the same size. The splenic dulness, together with palpation of the spleen in its normal position, settles the matter. The fecal masses may be removed by purgatives. A floating spleen may be enlarged and cause trouble in diagnosis. Additional spleens may often be present, but are rarely large enough to palpate. It may be extremely difficult at times to tell a floating spleen from a tumor of the kidney.

### **PRIMARY SPLENOMEGALY.**

This is a rare form of enlargement of the spleen, first described by Jaucher, which comes on without any apparent cause. The changes in the spleen consist in a hyperplasia of the endothelial cells. There may also be changes in the retroperitoneal and mesenteric lymph nodes and an increase in the connective tissue in the liver. The disease begins in early childhood, from the second to the seventh year, and there are slow but progressive changes. The enlargement of the liver is always secondary to the splenic enlargement, and never to the same extent. In addition there is a simple anemia, softening of the gums with oozing of blood, epistaxis, subcutaneous hemorrhages, and occasionally icterus. The symptoms are those referable to the splenic enlargement. There is pain in the abdomen, disturbances of the functions of stomach and intestines and sometimes dysuria. There may be

dyspnea. Cramps in the legs have been noted. The disease may last for years, the spleen eventually practically filling the abdomen. Bovard<sup>1</sup> has reported cases and discussed the subject fully.

#### NEW-GROWTHS.

These are rare in the spleen during early life, and may be difficult to differentiate from simple hypertrophy. If the spleen has an uneven surface, especially if there are prominent nodules, a new-growth may be present. This may be:

(a) *Tuberculosis*. Where there is tuberculosis elsewhere in the body. The commonest form of nodular spleen in children.

(b) *Sarcoma* may be primary or occur as metastasis. A rare condition.

(c) *Carcinoma* is very rare, but has been reported. Carcinoma is found elsewhere in the body.

(d) *Syphilitic irregularities* disappear or improve on treatment, and there are usually other evidences of the disease.

(e) *Cystic tumors* may result from hemorrhage and are of great rarity in children. They contain cholesterol, lecithin, and blood-coloring matter.

(f) *Parasites* (echinococcus) have been reported in Europe, but not in America to my knowledge.

<sup>1</sup> American Journal of the Medical Sciences, October, 1900.



## CHAPTER XXXIV.

### THE ADRENALS—ADDISON'S DISEASE—CRETINISM—DIABETES MELLITUS.

#### THE ADRENALS.

THE study of the adrenals has been much neglected in infancy. In early life they are relatively larger than in adults. In infants dying with severe general congestion they are markedly enlarged and may contain hemorrhages. In the reverse type, where the infants seem bloodless and the tissues anemic, the adrenals are small and contracted.

**Hemorrhage into the Adrenal.**—This occurs possibly as a separate disease.<sup>1</sup> Arnaud has described three classes of symptoms occurring with hemorrhage into the adrenal: asthenic, peritoneal, and nervous. It occurs in the course of gastroenteric infection; it is common in the newborn; it may occur in any disease where there is stagnation of the blood or congestion, as in acute or chronic diseases of the lungs, heart disease, and convulsions. It may be seen in septicemia, pyemia, acute miliary tuberculosis, and the various toxemias. Congenital syphilis has also been mentioned, but seems unimportant. Traumatism may be a cause and the so-called blood diseases, as scurvy, may play a part.

The whole gland may be transformed into a blood sac with extravasation into the surrounding tissue. There may be hemorrhage into the medulla of the gland, while the cortex remains free or nearly so, as there may be scattered hemorrhages into the gland substance, chiefly in the medulla.

**Symptomatology.**—There may be symptoms of an acute infection. In this there is an acute onset, generally in a previously healthy infant. There may be vomiting, diarrhea, and in a few hours a petechial or purpuric eruption may appear over the child. There is usually a temperature of from 101° to 105° F. The child collapses and dies. The diagnosis is usually made of one of the eruptive fevers. Many cases have been in unvaccinated children and have been called smallpox. (See *Purpura Fulminans*, p. 831.)

A second class of cases is seen where there is purpuric rash, but where there is nothing to suggest an acute specific fever. A third class cannot be recognized clinically, occurring as it does in the course of some pre-existing disease, as in pneumonia. This is only to be made clear at autopsy.

Lastly, hemorrhages in the adrenal are found in the hemorrhagic diseases of the newborn.

<sup>1</sup> Dudgeon, *American Journal of the Medical Sciences*, February, 1904.

**ADDISON'S DISEASE.**

Addison's Disease is characterized in children by the same bronzing of the skin and progressive cachexia that are found in adults. The lesion in nearly all cases is a tuberculosis of the adrenal, but a few instances have been reported where the lesion was a malignant growth in the gland. It has also been noted that there may be tuberculous lesions of the adrenal without any symptoms. There are usually tuberculous deposits elsewhere in the body, the lungs and the lymph nodes being most frequently affected. There are changes in the abdominal sympathetic nerves. The pigmentation of the skin is due to deposits of pigment in the Malpighian layer.

The disease becomes rarer the younger the age of the child. Under five years of age it has been seen occasionally, but is almost unknown. A congenital case has been reported. After eleven it is seen more frequently. Boys are affected slightly oftener than girls. In 21 cases collected by Comby, 12 were in boys and 9 in girls.

**Symptomatology.**—The onset is usually gradual. There may or may not be tuberculosis of lungs, lymph nodes, or of other organs. There is progressive weakness, with symptoms of stomach trouble. There may be vomiting and, in some cases, diarrhea. In some cases lumbar pains or vague pains in the limbs are complained of. There may be colic or headaches. As the disease progresses the patient becomes cachectic. The most marked thing is the pigmentation of the skin. This may be partial or general. The color is a dirty yellow, which becomes darker. The exposed parts of the body, face and hands, and the parts containing pigment, such as the areola of the breasts, the external genitalia, the groins, and axillæ, may be very dark or even almost black in color. The hair may change color. There are usually pigmented patches in the mucous membranes. The patient becomes weaker and weaker, and fever may develop. The pulse becomes rapid and filiform and the respirations are increased. Many of the patients die of tuberculosis of the lungs, others from asthenia. At the time of death there may be convulsions, coma, or syncope.

**Diagnosis.**—This is, as a rule, not difficult if the case is well developed. The symptom-complex of progressive weakness, pigmentation of the skin, the weak and rapid pulse, and the digestive disturbances serve to differentiate it from other conditions. One should bear in mind the pigmentation from arsenic. In this the use of the drug can generally be elicited. Malarial cachexia may be distinguished by the history of exposure, the parasite in the blood in many cases, the enlarged spleen and the effect of quinine. The presence of bile in the urine serves to differentiate icterus. The bronzing from exposure may resemble it quite closely as to the arrangement of the color of the skin, but the general health is usually good.

**Prognosis** is always bad. There have been some cases reported as cured, but with our present means of treatment this is not to be expected.

**Treatment.**—This consists in good general hygiene, proper care, and regulation of the diet. Cod-liver oil is most highly recommended for its nutritive value. Adrenalin may be tried. Of the 1:1000 solution from 0.06 to 0.03 c.c. (1 to 5 min.) or even more may be given. It has not been used long enough to state anything about the results. Feeding with adrenals may be tried in place of the adrenalin if desired. The glands may be given raw or nearly so, on bread or toast. A glycerin extract may be used. Tablets of the dried gland are also sold. One gland may be given once, twice, or three times a day. From one-quarter to a whole tablet may be given at a time. The effect in all cases should be carefully watched, and the dose regulated accordingly.

### CRETINISM.

Cretinism is a "chronic affection characterized by disturbance of growth of the skeleton and soft parts, a remarkable retardation of development, an extraordinary disproportion between the different parts of the body, a retention of the infantile state, with a corresponding lack of mental progress."

Endemic cretinism has been known for a long while. In certain mountainous, limestone districts, as in parts of Switzerland, there are frequently seen peculiar dwarfs, of short stature, short arms and legs, with a myxedematous condition of the subcutaneous tissue. The mentality is exceedingly low. In a rather large percentage (60 per cent.) there is a goitre. Most of these endemic cretins die before thirty.

Sporadic cretinism is a similar condition met with all over the world. The cause is unknown. The cretin is an individual whose growth has been retarded. Mentally they are idiots and physically dwarfs, with the characteristics described below.

Usually there is only one cretin in a family, the other children being perfectly normal. In some instances there have been more than one in the same family.

**Pathology.**—The condition is due to a lack of or insufficiency of the internal secretion of the thyroid gland. There may be an absence of the thyroid, an atrophy of it, or there may be a goitre. This last is not very frequent in sporadic cretinism. In Osler's 60 cases it was present in 7. The changes may be congenital or may develop after an acute infectious disease. Cases have followed measles and typhoid fever. In these there is an atrophy of the thyroid, apparently due to some poison produced by the acute infection.

There is a lack of development on the part of the entire body. The child is dwarfed. The ossification of the bones goes on very slowly and imperfectly. There is in the subcutaneous tissue a substance giving the reaction of mucin which causes the edematous appearance; hence the name myxedema which is given to the case occurring in adult life.

An interesting class of cases seen in older individuals is that following the operation for the removal of the thyroid. Where this has been

complete a myxedematous condition has supervened. A small part of the thyroid left behind will prevent this. The acute symptoms coming on a few days after operations, consisting of tetany-like convulsions, great prostration, and death, are due to the removal of the parathyroids, small glandular bodies near or on the thyroid which evidently play an important part in the animal economy.

Myxedema can be produced experimentally in animals by removing the thyroids. Sporadic cretinism may be regarded as infantile or juvenile myxedema.

In some instances the function of the gland, while impaired, is not entirely destroyed. This may give rise to symptoms which have the appearance of mild myxedema, and these have been described by French and Belgian writers as *myxodème fruste*.

**Symptomatology.**—The symptoms may come on at any time. Cases have been noted a few weeks after birth. In others about the first or second year. These may have escaped notice until a lack of development calls attention to the condition. Other cases may appear later and these either follow some acute infectious disease or some unknown cause.

The cases seen in early infancy may be difficult to recognize unless they are very pronounced. The infant is sluggish and torpid and does not pay attention to anything. The temperature is below normal and the body is easily chilled. It feels cold to the touch. The expression is not suggestive of healthy infancy. The eyes are puffy and the tongue may protrude through the open mouth. The tongue itself is thick and unshapely. The cry is hoarse and guttural. The abdomen is prominent. As time goes on these characteristics become more and more pronounced.

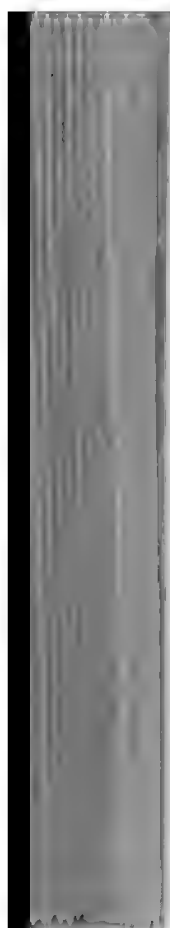
The cases seen about two years of age may be told at a glance. They have a very characteristic appearance. They are shorter than normal children of the same age. The body is proportionately larger than the extremities, and the head seems too large for both. The forehead is low and the fontanels open. The fontanels may remain open until ten or twelve years of age or even later than that. The hair is coarse and straight. The face is very striking and well illustrated in Plate XXVI. The expression is pig-like. The base of the nose is broad and the eyes wide apart. The eyes are slit-like, reminding one of pigs' eyes; the eyelids are puffy. The eyebrows are scanty or wanting. The cheeks are large and sag. The lips are prominent; the tongue is thick and usually protrudes through the half-open mouth. There is a tendency to be drooling. The teeth are erupted late and are apt to decay early. The neck is short and the head seems set directly on the trunk. The arms and legs are short and misshapen. The patients assume a squat attitude and generally have more or less kyphosis or lordosis. The hands are short and spade-like, with broad, prominent hypopharyngeal eminences. The genitalia are edematous-looking and remain undeveloped throughout life in the untreated cases. The abdomen is prominent and pendulous. The skin is coarse and rough, sallow and waxy. There is a tendency to eczematous skin eruptions. The cut-



PLATE XXVI.



Sporadic Cretinism. Child fifteen months of age. (Koplik.)



body has an edematous appearance, but there is no pitting on pressure. The thyroid may be absent and there may even be a depression in its place. In the older cases there may be subcutaneous fatty tumors which are usually symmetrical and most frequently just above the clavicles or above the shoulders. Many cretins are deaf-mutes, but if they talk the voice is hoarse and guttural. There is usually marked constipation. If the cretin walks at all he is late in learning, and he may be five or six years old before he makes any effort. When he does walk the gait is uncertain and of a waddling character. Cretins are sluggish, lethargic individuals, who lead a rather vegetable type of existence. They may have epileptiform seizures.

As time progresses all these features become more pronounced. They remain short and undersized, and when the disease has begun in early infancy a cretin of twenty may have the appearance of a child of three or four, or even younger. Their mental development is about equal to that of children whose age they resemble. They talk but little, if at all, and are child-like in all particulars. I know of a cretin of fifty-eight, the size of a small girl, who still sits on the floor and plays with her dolls.

The partially developed cases, the *myxœdème fruste* of the French, may be less easy to recognize. A child late in teething, with an open fontanel, who ceases "to get on," should always suggest cretinism. The skin becomes lax, the child gets fat and flabby, and the abdomen prominent. The cretin appearance may be more or less marked. The loss of vivacity is striking.

**Diagnosis.**—This is easy as a rule. A child with an open fontanel later than eighteen months, the delayed dentition or any of the other features described, should suggest the disease. Once having seen a case, or a photograph of one, it is difficult to mistake a well-developed case of cretinism. The differential diagnosis is from several other conditions.

**MONGOLIAN IDIOCY.**—This most nearly resembles cretinism. There is a distinct Mongolian type of face; they are dwarfed and of a low grade mentally. These cases are, as a rule, much more sprightly than cretins. They have no myxedematous condition of the subcutaneous tissues and are more shapely than the cretin. The hands may show a crooking of the little finger.

**INFANTILISM.**—Mild grades of cretinism might be confused with infantilism, but not marked cases. Infantilism is a "morphological syndrome characterized by the preservation in the adult of the exterior form of infancy with the non-appearance of the secondary sexual characters." The following is a translation of a French description of the condition (Lamy): "The face is rounded and chubby, the lips prominent and plump, the nose poorly developed, the face smooth, the skin fine and of a clear color, the hair fine, the eyebrows and lashes sparse. The trunk is long and cylindrical. The abdomen is somewhat prominent, the arms and legs plump and tapering from the trunk to the extremity. A layer of adipose tissue surrounds the body and masks

## RES OF BLOOD, LYMPHATIC SYSTEM AND

and muscular prominences. The genital organs are rudimentary. There is an absence of hair on the pubes and axilla. The larynx is poorly developed and the voice is shrill and piercing. The larynx is poorly developed and the voice is shrill and piercing. The larynx is poorly developed and the voice is shrill and piercing.

"Infantilism may be seen in hereditary syphilis. The delayed dentition and the open fontanel might lead to a diagnosis by a careless observer, but the rachitic child is much more robust and the skin is more often moist than dry (see p. 321).

**ACHONDROPLASIA.**—This is a curious form of dwarfism, called also achondroplasia, usually congenital, but exceptionally appearing a few years after birth. The majority of cases are born dead or die soon after birth, and very few reach maturity. They are frequently seen in the form of Fetal Rickets or Fetal Myxedema. P. Marie called attention to a dystrophy of the epiphyseal cartilages. Pathologically the disease is usually easily recognized. These patients have very large heads and very short arms and legs (Figs. 173, 174 and 175). The humerus and femur are apt to be quite short. The trunk and thorax, while small, are normal. The long bones show considerable hypertrophy at the epiphyses, but the shafts of the bones are normal. The hands are peculiar, short, and spade-like. They have been called "trident shaped," from the deviation of the last two phalanges. The first phalanges are close together, and the last two phalanges. The first phalanges are close together, and the last two phalanges.

The intellect is usually about that of children of the same height and they are exceedingly mischievous. Sometimes they may have fair minds. Unlike other dwarfs, they are well developed sexually and have strong sexual instincts. They are sometimes mistaken for cretins, with whom they have nothing in common, but are easily distinguished by the above-mentioned points. From rickets the points of diagnosis are apparent, and the two diseases are not associated.

**Prognosis.**—The prognosis in untreated cases of cretinism is bad. They remain hopeless idiots. Death generally takes place from some intercurrent affection before thirty years of age, but occasionally they live much longer. With treatment the outlook is very good in all cases seen young. After puberty the results, while fairly satisfactory, are not nearly so brilliant. After adult age has been attained comparatively little benefit is derived from treatment, but even then the results may at times be striking. This, of course, applies only to cretins who have been so from infancy and not to cases of myxedema acquired in later life. In these latter cases the results of treatment are very satisfactory.

**Treatment.**—The treatment of cretinism is one of the most brilliant results of modern medicine. The credit belongs to a large number of workers, chief among whom may be mentioned the physicians Goetz and Ord, who described the adult type of myxedema, the surgeons Kocher and the Reverdins for experimental and operative work, and the physiologists Schiff, Horsley, and von Eiselsberg for the direct demonstrations of the possibilities of treatment. If thyroid gland supplied to the body, the effect is wonderful. Experimentally the living



gland was first grafted into the body; later the patients were fed on the fresh glands, and then the dried gland was used as being more convenient for administration. The desiccated thyroids are now supplied

FIG. 173



FIG. 174



Achondroplasia (chondrodystrophy).

in tablet form by several manufacturing chemists. The tablets each represent 0.324 gm. (5 gr.) of the fresh gland of the sheep. The dose should be small at first and gradually increased. For infants it is well to begin with a quarter of a tablet, three times a day. If no effect is noted the dose may be gradually raised to 0.324 gm. (5 gr.) three times

a day, and when this is not effective two or even three tablets may be given as a dose. If the dosage is too great unpleasant symptoms occur;

FIG. 175



Achondroplasia. (Case of Drs. West and Piper, Archives of Pediatrics.)

fever, rapid pulse, and flushing are the principal ones. Should these occur the dose should be diminished. It is well to break the treatment occasionally and give the patient a few days' rest.

The effect of the treatment is marvellous. After a month or six weeks' time there is a loss of weight and the myxedematous appearance gradually disappears. The expression becomes more natural, the face loses its puffy appearance, the palpebral orifices become wider, the abdomen decreases in size, and the child's figure assumes the shape of a normal child. The hair and skin become more natural in appearance. In younger infants teething goes on rapidly. In older individuals in whom the milk teeth have not been shed there is a replacement of these by the permanent teeth. The growth in height is very striking: from four to eight inches, and even more, has been noted in a year. The mental change is also marked. The change is greatest in young cretins, but the older ones may also be benefited. The child begins to talk, and if it talked before it rapidly acquires larger vocabulary. The whole being transformed from the condition of a vegetable to that of a living human being.

The treatment should be continued until all traces of the myxedematous condition have disappeared, and until natural growth has been established. After that time very small doses should be continued throughout life, one or two 5-grain tablets a week seem to be sufficient to keep the individual in good condition. This should be insisted upon when taking charge of a case. The treatment may be stopped for a month or six weeks, but if it is discontinued for any longer the symptoms begin to return. The child becomes listless and begins to show other symptoms of a return of the trouble.

#### DIABETES MELLITUS.

Diabetes Mellitus is characterized in children by the same symptoms as seen in adults, the most notable being the glycosuria, polyuria,

increased appetite, increased thirst, and the progressive loss of weight. It must be borne in mind that diabetes mellitus is manifested by a symptom-complex, and that the mere presence of sugar in the urine may not mean diabetes.

The disease is rare in childhood, but probably not so rare as was formerly supposed. Owing to the carelessness about examining the urine of young children, and the extreme difficulty in securing it in some instances, the disease may easily be overlooked.

It is a difficult disease to study in children, and our knowledge of the subject is based on comparatively few cases and on fewer autopsies.

**Etiology. Frequency.**—West, in 700 cases, gives only 1 under five years. Ashby and Wright mention 111 from six months to fifteen years of age. Senator found 1 case in 5900 children applying at a polyclinic. Ebstein found 1 in 694 children. Pavy in 1360 cases of diabetes gives 8 under ten years. Seegen in 800 cases of diabetes gives 4 under ten years. Schmitz in 600 cases gives 5 under ten years.

**Age.**—Orloff mentions 7 cases in nursing infants. Undoubted cases have been reported as early as four months of age. The very early cases, from fourteen days to one month, are supposed to be lactosuria, as they recovered.

	Wegeli.	Leroux.
0 to 1 year . . . . .	1 case.	1 case.
1 to 5 years . . . . .	26 cases.	23 cases.
5 to 10 " . . . . .	81 "	43 "
10 to 15 " . . . . .	42 "	71 "

**Sex.**—In adults the males preponderate. In children the sex influence seems very slight. Males are slightly more often affected before five years, but from five to fifteen years the sexes are about equal, or if anything a slightly larger number of females are affected.

Diabetes is frequent in Jews, in adults, but race influence apparently plays but little part in children.

**Heredity.**—There is a strong hereditary tendency to the disease. Many instances are on record where there are two or more cases of diabetes in the child's ancestors or family. Of the other diseases mentioned the neuropathic tendency and gout are the most frequent. Syphilis and rheumatism are also given as predisposing causes.

**Exciting Causes.**—Blows or injuries to the head are given as a frequent exciting cause. Wegeli mentions 11 out of 108 cases where the diabetes followed trauma of the head. Blows on other parts of the body are also mentioned, especially the spine, back, and abdomen. Various nervous diseases, such as tuberculous meningitis, chorea, and epilepsy are sometimes associated. Cold is also reckoned as a causative factor. The influence of previous diseases, especially the infections, must not be forgotten. Too much sugar and starch in the food, particularly when there is an hereditary predisposition, may apparently bring on diabetes. Starch indigestion may be noted in some children with an hereditary tendency to diabetes.

**Pathology.**—This is apparently the same as in adults. As yet the subject is not at all clear. The morbid anatomy is based on compara-

tively few autopsies. Lesions have been noted in the floor of the fourth ventricle and also in the pancreas.

**Symptomatology.**—This is the same as in the adult. There are polyuria, glycosuria, increased appetite, and increased thirst.

The polyuria is quite constant. The child may require changing twenty or thirty times a day. When the urine can be measured it varies between 1 and 5 litres, although cases have been reported where in twenty-four hours the child passed the remarkable amount of 16 litres. The polyuria is more marked during the day than at night. Enuresis is a frequent symptom and one which should always lead to an examination of the urine. Coming on in a child who has previously held his urine all night it is suggestive.

The appetite is usually very much increased, and gastric and intestinal disturbances are frequent. Diabetic children are usually constipated. The gums are frequently swollen and bleed easily. The mouth and tongue are usually dry.

The thirst is very pronounced, and the children drink or attempt to drink any fluid in sight.

The skin becomes dry and scaly and there are frequent eczematous diseases or furunculosis. Pruritus of the genitalia is common. Edema may be seen occasionally.

The children waste away, and if the disease lasts any length of time they become veritable skeletons. They lose strength as well.

Headache is frequent and they complain of other pains and neuralgias. The patellar reflexes may be diminished or lost altogether. There is an alteration in the child's character. Diabetic children become irritable, cross, capricious, and later on they may become apathetic. Insomnia is usually present.

The sight may be diminished almost to blindness. Von Graefe and others have reported diabetic cataracts in children.

The disease frequently comes on suddenly in young people and generally runs a rapid course. As a rule, it is a question of weeks or months. Cases have been reported where death ensued twelve days after the sudden onset. Külz gives 46 cases as follows: 16 lived less than three months, 14 less than one year, and the others lived between one and four years. Death usually takes place from pneumonia, tuberculosis, asthenia, or coma.

**Diabetic Coma.**—There may be prodromes, consisting of increased feebleness, a sweetish chloroform-like odor of the breath, and diacetic acid in the urine. The attack may come on with vomiting and diarrhea. The child becomes apathetic and soon loses consciousness. The pupils are fixed and equal and may be either dilated or contracted. The knee-jerks are abolished. The rectal temperature is generally lowered, although it may be raised. The pulse is rapid and the breathing irregular. It is usually of a deep, sighing character, and may be of the Cheyne-Stokes type. Sometimes it is called dyspneic coma.

The child becomes algid and cyanotic and generally dies in from eighteen to thirty-six hours.



**The Urine in Diabetes.**—This is practically the same as in adults. The color is pale, the specific gravity raised to 1.030 or 1.040, but this may vary greatly. Proportionately the amount of glycogen excreted is rather greater in children than in adults. There may be albuminuria. There may be acetonuria, especially a few weeks before death, and there may be diacetic acid in the urine.

**Diagnosis.**—This is made as in adults, on the presence of the cardinal symptoms and by the examination of the urine. The fermentation test is the most reliable.

Care should be taken to exclude lactosuria, which is occasionally seen in infants. Alimentary glycosuria depending on the ingestion of large amounts of sugar should be excluded by cutting down the sugar in the diet to a minimum.

By the following test Bremer claims to be able to make the diagnosis of diabetes, not only when there is sugar in the urine, but also during the sugar-free intervals. Moderately thick smears of blood are made on the glass slides. A smear of normal blood is made as a control. These are heated in a thermostat up to 135° C. When cooled they are placed back to back in a tall staining dish and stained two minutes in a 1 per cent. solution of Congo red. The stain is washed off. Normal blood takes a red stain. The blood of diabetics does not stain at all by this method.

**Prognosis.**—The prognosis is very grave. Death is the rule. Senator says that no form of treatment is of any use. In 28 cases of Wegeli's, all personal observation, 23 died and 5 still had diabetes at the time of the report. The reported cures are usually of alimentary glycosuria or lactosuria, or are only remissions in the real disease, which appears later if the case is followed.

**Treatment.**—This is of little avail, but may be tried, as von Noorden says that we do not know what strict diabetic treatment might accomplish, since it is seldom tried in children.

Infants may be allowed to nurse at the breast. Vichy water is advised in these nursing infants by French writers. A teaspoonful is given at each nursing.

Bottle-fed babies should be fed on modified milk, sweetened with saccharin or mannite, and may be given egg-water or beef-juice and broths as well. Cream should be given in as large quantities as possible.

Infants from one to three years may be given a litre of milk a day, with cream, egg-water, meat-juices, broths, raw scraped beef, and purees of green vegetables (as of peas) given in addition. The usual diabetic breads may be tried.

In older children the diet should be along the same lines as that recommended for adults. The main indications are to keep up the patient's strength, and, if possible, to increase it by giving food which can be utilized by the body and to avoid complications. These indications are best fulfilled by a diet consisting of a carefully balanced mixture of proteids and fats, with as little starchy food as possible and no sugar.

In severe cases von Noorden's oatmeal cure may be tried.<sup>1</sup> This consists in giving a very well cooked oatmeal to which vegetable albumins or egg-albumen and butter have been added. It is given every two hours, and coffee and some form of alcoholic beverage may be allowed. Every week or ten days meat and vegetables are allowed for a day to break the monotony of the diet. Return to an ordinary diet must be gradually made. Remarkable results are said to be obtained by this diet in severe cases. It is not useful in the lighter forms.

*Prophylactic Diet.*—In diabetic families it is, perhaps, a good thing to limit the amount of carbohydrate food. It is a question whether this has any effect in diminishing the probability of the individual's developing diabetes. Good, sensible, hygienic living should be insisted upon, and any tendency to obesity managed by diet and exercise.

*Medicinal Treatment.*—A great many drugs have been advised. Opium, morphine or, preferably, codeine, give the best results. Bromide of potassium is also of value in some cases and antipyrin useful in the extremely nervous patients. Arsenic has been recommended, as has nearly every drug in the pharmacopeia. Lactophosphate of lime has recently been used by some observers with reputed success.

<sup>1</sup> Friedenwald and Ehrlich. *American Journal of the Medical Sciences*, October, 1905.

# SECTION XI.

## DISEASES OF THE NERVOUS SYSTEM.

By D. J. McCARTHY, M.D.

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### CHAPTER XXXV.

#### FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM--CONVULSIVE DISORDERS.

**Methods of Examination.**—The elicitation of symptoms referable to the nervous system in children, and especially in infants, is usually associated with some difficulty. As a general rule, it is necessary to have the child thoroughly stripped. Any palsy, atrophy, or deformity becomes apparent to the eye of the examiner. For the determination of reflexes it is also quite necessary that the child should be stripped and as far as possible relaxed. It is to be remembered in this connection that in the *Babinski reflex* extension, instead of flexion of the toes, when the foot is tickled, a pathological condition in the adult and even in the child after it has begun to walk, is a normal phenomenon in the infant.

**Reflexes.**—The normal *plantar reflex* of the walking child, as in the adult, is a marked flexion and adduction of the toes when the plantar surface of the foot is irritated by slowly drawing a blunt point along the outer or inner surface of the sole. The ankle should be firmly held by the left hand and the knee should be flexed before attempting to elicit this symptom. The other reflexes of importance to be considered in the diagnosis of diseases of the nervous system in children are as follows:

**The Knee-jerk.**—This is obtained in young children only with difficulty. It is quite necessary to have the patellar tendon in a condition of slight tension and to have the attention of the child distracted, and even then in some children it is only after repeated efforts that this phenomenon can be demonstrated.

**The Achilles-jerk.**—This is best obtained by having the child in a kneeling position and then giving a slight tap with the percussion ham-

mer at the insertion of the Achilles tendon. This reflex is usually obtained without much difficulty.

*The Biceps-jerk.*—The arm of the child should be held relaxed and in a flexed position in the arm of the examiner. The thumb is placed over the biceps tendon and a slight tap of the hammer on the thumb produces a reaction in the biceps which can be easily felt by the palpating thumb.

*The Triceps-jerk.*—The arm should be flexed and allowed to hang loose over the arm of the examiner; a slight tap is then given at the insertion of the triceps tendon.

*The Chin-jerk.*—This is obtained by a slight tap directly on the chin or on a finger of the examiner held against the relaxed chin; a sudden jerk of the jaw is frequently but not always obtained.

*The Cremasteric-jerk.*—Scratching the inner surface of the thigh results in a contraction of the scrotum and elevation of the testicles.

*The Superficial Abdominal Reflexes.*—Scratching the skin of the abdomen below the lower margin of the chest results in a sudden muscular contraction on the side irritated.

*The Eye Reflexes.*—The reactions of the pupil to light and to accommodation are obtained in the usual manner.

*The Examination for Sensation.*—The determination of disturbances of sensation is obtained, first, by watching the expression of the child's face when a comparative test of the application of a pinpoint is made on the two sides of the body or between an area of normal sensation and the affected area; second, by the degree of muscular retraction after the application of the above tests. In transverse lesions of the cord the upper limit of sensation is easily determined by drawing a sharp point along the surface of the skin, beginning in the area of loss of sensation, and noting the evidence of painful impression when the normal skin is approached.

*The Electric Examination.*—In making an examination of the muscles for reactions of degeneration it is usually necessary in young children to make use of the electrodes without current so as to accustom and reassure the child to the use of the apparatus. In cases where it is impossible to determine the formula of electric degeneration, the character of the muscular reaction itself is as valuable in determining the presence of degenerative atrophy. The normal muscle reacts with a quick, lightning-like contraction; the degenerating muscle gives a slow, vermicular reaction in proportion to the extent of the degeneration.

*The Motor Power.*—Marked loss of motor power is manifest on inspection by the position of the part affected, the flaccidity of the muscles, and, in the case of the upper extremity, the use by preference of the opposite limb in simple motor acts.

*Lumbar puncture* and its diagnostic value are considered under Tuberculous Meningitis, p. 382



## FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM.

## CHOREIFORM DISEASES.

There is an extensive group of disorders of motion occurring in childhood, and not infrequently extending into adult life, to which the name of Chorea has been given. Among these diseases we recognize the following:

1. Acute Chorea. Chorea Minor.
2. Chorea Major.
3. Habit Chorea. Habit Spasm.
4. Electric Chorea.
5. Chronic Progressive Chorea. Huntingdon's Chorea.
6. Organic Chorea. Post-hemiplegic Chorea. Athetosis.

**Acute Chorea.**—This disease, also called Sydenham's Chorea, Chorea Minor and Saint Vitus' Dance, is the most important in our classification. It is almost entirely confined to childhood and characterized by irregular, involuntary, purposeless movements affecting the voluntary muscles.

**Etiology.**—It occurs with much more frequency in the female sex and is especially prone to affect those of a nervous temperament. It is rare in infancy and after puberty. The largest number of cases occur between the fifth and fifteenth year. Girls of the lower classes, of poor nutrition, and subject to the strain of public-school education and the worry of examinations furnish a large percentage of the cases studied. If the infectious fevers rheumatism plays an important part in the etiology of this infection. The acute articular inflammatory type of rheumatism as a cause of chorea is comparatively rare. This has occurred in only two cases of the last sixty cases coming under my observation. Vague pains about the joints, with occasionally a history of swelling and tenderness, occur much more frequently. There is still a third group of cases presenting, without articular or pain manifestations, an acute or subacute tonsillitis or pharyngitis. If all the above manifestations be classed as rheumatic, we are necessarily forced to consider the relation between rheumatism and chorea as very close. The French writers contend that there is a very close relationship between rheumatism and chorea; the German school on the other hand, while admitting a certain relationship, do not go so far as the former. The American authorities have sometimes taken the one position and sometimes the other. The clinician of internal medicine is more likely to describe a close casual relationship between rheumatism and chorea than the specialist on nervous diseases, because the physician is more likely to see the chorea as a complication of rheumatism than to see the isolated case of chorea, and to investigate the relative frequency of rheumatism, as a cause. Poynton states that chorea is "in most instances, if not in all, rheumatic origin." Of the five hundred and fifty-four cases studied by Osler, fifteen and five-tenths per cent. gave the history of rheuma-

tism in the family; fifteen and eight-tenths per cent. gave a history of arthritic swelling, acute or subacute, and only twenty-one per cent. as a maximum gave a history of rheumatism or vague pains sometimes described as rheumatic in various parts, but not associated with joint trouble. Kaufman, in a study of forty cases, gave rheumatism as a complication in six of these. As an example of what these statistics mean, however, in one of the cases the rheumatism antedated the chorea three years. The following table states the relationship in two columns. In the first column the percentage is that of an antecedent rheumatism. In the second column of a coincidental rheumatism complicating the chorea.

	Per cent.	Per cent.
1. Sturges. . . . .	26.5	1.45
2. Dickinson . . . . .	26.75	7
3. Pencock . . . . .	28	7.66
4. Owen . . . . .	26	8
5. Ogle . . . . .	...	10

In many of the articles written upon these subjects, the term rheumatism is used in such a general way to express vague pains, headache, etc., that they are of little value in studying the true relationship between rheumatism and chorea. I stated above the presence of acute inflammatory articular rheumatism was only two cases in sixty. These were the only two cases which presented a clinical picture of undoubted acute articular rheumatism. Osler's masterly study of the subject is of most value in considering the etiology of the disease. Among the other acute infections scarlet fever and acute pyemia occasionally antedate chorea.

As adult life is approached pregnancy may become an important factor. Among the determining factors fright and mental emotion have long been considered of much importance. A careful study, however, of a large number of cases leads me to the conclusion that they have little if any direct influence in the production of the disease.

**Pathology.**—Many minor conditions of the central nervous system have been found. To none of these, however, can the symptoms with any degree of certainty be ascribed. Hyaline degeneration of the vessels, perivascular leukocytic infiltration, capillary hemorrhages, cell degeneration, and thrombosis of the cerebral capillaries have been described in a very small number of cases. The fact that all of these conditions occur not infrequently without the production of chorea movements forces us to consider this disease as a functional disorder of the motor cortex. This is also borne out by the psychic manifestations. The presence of emboli of the cortical vessels and of embolism of the central artery of the retina in a small number of cases may be considered as a result of the complicating endocarditis rather than as a cause of the disease.

**Symptomatology.**—Cases of chorea divide themselves naturally into three groups according to the severity of the affection: (1) mild chorea, (2) severe chorea, and (3) malignant chorea, or chorea insaniens.

*Mild Chorea.*—After a week or so of depression and nervous irritability slight, irregular, purposeless movements are noticed in one of the upper extremities. This is associated with a pseudo loss of power. There is a tendency in nearly all cases to drop articles, such as dishes, spoons, etc., even before the parents notice the irregular movements. In the fully developed disease this inability to hold articles is associated with the involuntary movements. At the beginning of a choreic jerk of the arm the hand is also affected, and a relaxation of the grasp occurs simultaneous with the movement. The movements extend at first to the lower extremity of the same side and may be confined to one side of the body throughout the disease. In the greater number of cases the disease extends to the opposite side and to the face until all the voluntary muscles are affected. The movements of the mild form of the disease cease during sleep. The tongue may be affected and the speech become jerky and mumbling in character.

In a small proportion of cases, independent of the intensity of active motor disturbance, a real loss of power occurs, usually hemiplegic in distribution, called *post-chorea hemiplegia*. The loss of power never amounts to a complete paralysis, but may be sufficiently intense to produce difficulty in walking and an inability to raise the arm above the level of the shoulder. In a case recently under my observation the hemiplegia was so marked that the boy was compelled to drag the leg in walking, and was not only not able to raise the arm, but was unable to hold it in an elevated position after it had been placed there. In this case the choreic movements were only slightly marked and confined at first to the palsied side, but later extended to the rest of the body. The weakness of the right side rapidly disappeared under treatment.

The mentality is affected even in mild cases. There is a dull expression of the face and marked irritability. Outbreaks of temper and marked emotional disturbances, such as crying spells, are frequent, and in a small number of cases, night terrors.

*Severe Chorea.*—The symptoms are practically the same as those of the mild form, differing only in intensity. The movements become much more marked and constant, the respirations become jerky and irregular, the heart action irregular, and the child speaks only with difficulty and with an accentuation of the movements, or at times is unable to speak at all. While the movements in this form are only discontinued during sleep they may in some cases be subdued, and the child may be awakened by a sudden jerky movement of an arm or leg. Motor weakness is in this form the rule, but whether it is a real loss of power or due to the associated movements is often difficult to determine. The mental symptoms are much more accentuated—the child lacks power of concentration, is very irascible, and has failure of memory. There are cases on record of distinct mental alienation, melancholia, dementia, etc., but no such cases have come under my own observation. Fever is present in a large number of cases. It is usually very slight, 1° to 2°, but at times may be as high as 101° F. A decided temperature should always suggest the possibility of some complication.

*Malignant Chorea or Chorea Insaniens.*—This may be a terminal condition of the severe form or may develop as a distinct type from the beginning. It occurs more frequently as we approach adult life, and there is usually some source of mental worry or intense anxiety as a complicating factor in the etiology. The motor manifestations become intense, universal, and constant. They interfere with the sleep of the patient, and rapid exhaustion occurs. A confused delirium or a wild maniacal outbreak ensues, the temperature rising as high as 104° F., and a fatal termination is the usual result.

*Complications.*—Conditions usually considered rheumatic in character are the most frequent complications. The most important of these are erythema nodosum, subcutaneous rheumatic nodules, rheumatic purpura, and cardiac complications. (See p. 575.)

*Endocarditis.*—Many of the children affected are in such a poorly nourished and anemic condition that functional or accidental murmurs would naturally be expected. Care should therefore be exercised in differentiating functional murmurs, which occur with surprising frequency, from those due to an acute active endocarditis. It must be remembered that organic murmurs sometimes disappear. A soft systolic murmur heard along the base of the heart and even as far as the left sternal margin, with a normal outline of cardiac dulness, in a poorly nourished anemic child, may be considered functional, but requires observation. A rough or harsh murmur either with or without associated enlargement of the heart and displacement of the apex beat, and also heard in the axilla, indicates an active valvular endocarditis. Osler found in 72 of 140 patients examined, more than two years after the attack, evidence of organic heart disease.

*Pericarditis.*—In cases with distinct evidences of articular rheumatism pericarditis is an occasional complication.

Herpes zoster occasionally occurs, and may be attributed to the use of arsenic.

*Diagnosis.*—If the irregular, purposeless character of the movements of chorea be kept in mind there is little difficulty in making the diagnosis. Friedreich's ataxia presents slow, irregular, more or less atetoid movements, which may be mistaken for chorea. There is, however, a history of the disease affecting other members of the family. Nystagmus, a scanning speech, and loss of power are also present. The quick, jerky movements of chorea are altogether different from the movements of Friedreich's ataxia. The choreic movements of hysterical children simulate very closely at times those of true chorea; this is particularly so where the children have an opportunity to observe cases of true chorea. In the hysterical type the individual movements are exaggerated, usually rhythmical in type, and frequently disappear when the attention of the child is distracted, and lessened if not altogether absent when not under observation. There are usually other associated symptoms of hysteria: convulsions, anesthetics, contraction of the visual fields, reversal of the color fields, spasmodic strabismus, etc. The diagnosis can usually be made with certainty by the influence



of suggestion and hypnotism as they completely control the movements. It is to be remembered, however, in this connection that minor hysterical phenomena are frequently present in true chorea.

The motor weakness of true chorea may very easily be mistaken for an organic paralysis due to hemorrhage of the brain, cerebral embolism, poliomyelitis, etc. The history of the affection develops insidiously without disturbances of the reflexes and with the presence of the choreic movements which, while they at first may be minor, become more marked as the disease progresses. If the rule of diagnosis of nervous affections of childhood, of carefully inspecting the naked child before beginning the routine examination, be observed, this mistake in diagnosis will not occur.

The maniacal form of malignant chorea may with some difficulty be mistaken for Bell's mania, hysterical insanity, etc. Attention to the jerky, irregular, purposeless character of the movements will easily lead to a correct diagnosis.

**Treatment.**—The care and the prevention of functional disorders of the nervous system will be fully treated in a subsequent chapter. Choreia at all times, even in the milder forms of the affection, is sufficiently serious to demand the careful attention and supervision of the physician. All cases do better, the course of the disease is shortened, and the danger of complications lessened, by confining the child to bed during the period of active symptoms. Anything that tends to produce mental excitement should be rigidly excluded. A nurse trained to handle nervous conditions is a very helpful adjunct to the treatment. The diet should be simple and nutritious, with tea and coffee excluded. In the severe cases isolation is necessary. Care should always be used in permitting visitors; strangers should be excluded from the sick-room, and even members of the family when their visits or presence produce undue excitement. School duties and intellectual efforts should be avoided.

Gentle massage, with warm bathing and a warm or cold wet pack often have a quieting influence when properly administered. It is quite necessary in the poorly nourished, where overfeeding is necessary, to keep the muscles in good condition by routine massage, followed during convalescence by passive and resisted movements. These, however, should be carefully watched and, if any tendency to accentuation in the motor phenomena is manifested they should be decreased or stopped altogether. Electricity is sometimes of value both as a body stimulant and in keeping the muscles in good condition, but often produces too much excitement to be used. Galvanism should be applied in preference to faradism.

The medicinal treatment is confined to the use of alterative tonics and nerve sedatives. Of the former arsenic in the form of Fowler's solution is of distinct value. It should be given in small doses 0.12 to 0.3 c.c. (two to five drops) and increased by one drop a day until fifteen drops are reached. In older children it may be increased to 1.2 c.c. (twenty drops) three times a day. It is, however, inadvisable to give large doses in cases not under the direct observation of the physician. Pain or other

disturbance of the stomach with puffiness about the eyes should be the signal to stop the use of the drug. It may, however, be continued later. The long-continued use of arsenic may lead to arsenical neuritis. Care should therefore be used in prescribing this drug in dispensary cases.

Donovan's solution of iodide of arsenic and mercury, in doses of 0.1 to 0.3 c.c. (two to five drops) three times daily. Strychnine, cimicifuga, and belladonna may be of use. In cases where there is marked irritability, bromide of soda in combination with small doses of strychnine has a very sedative effect. The bromides and trional in doses of from 0.325 to 0.65 gm. (5 to 10 gr.) are of value in disturbed or restless sleep.

Inasmuch as recurrences of this affection are very common (as often as six to eight attacks in successive years being recorded) great care should be used in the hygienic surroundings of such children, and they should be removed from the overwork and excitement of the spring examinations, particularly at the public schools, at which time relapses frequently occur.

**Chorea Major.**—The Chorea Major (Epidemic Hysterical Chorea), the dancing epidemics of the Middle Ages, to which the terms Saint Vitus's dance, Saint Anthony's dance, etc., were originally applied, finds its prototype of the present day in epidemics of hysterical outbreaks among negroes at religious revivals. There is a condition occasionally met with in institutions for the care and education of children resembling true chorea and affecting large numbers of children. In the epidemic reported by Weir Mitchell at the Church Home for Children near Philadelphia there were, besides rhythmic choreic movements, hysterical convulsions, illusions, and hallucinations affecting a large number of children. The clinical picture was the same in all the cases, with slight modifications, and was suggested to the children affected by the attack of the first child. Epidemics of this affection are best treated by isolation of the individual cases, rest in bed, massage, overfeeding, and suggestion.

**Diagnosis.** The absence of fever, the normal condition of the reflexes, and the evident hysterical character of the affection will differentiate it from epidemic cerebrospinal meningitis.

**Habit Chorea.**—Habit Spasm or Convulsive Tic. This condition is very frequently met with in children from six to fourteen years of age, although it may occur at any time of childhood or adult life. In children of neurotic temperament, or those with an apparently normal nervous mechanism, but with defective home or school training, and in "spoiled children" who have never been subjected to proper discipline or training, habit spasms are of frequent occurrence. The movements differ from those of Sydenham's chorea by their evident purposeful character and their localization to a single muscle or a group of muscles.

**Symptomatology.**—The muscles of the face are most frequently affected. A sudden, quick blinking of the eyes, which may be repeated very frequently at short intervals, or occur only a few times during the

day, is the form most frequently met with. The eyebrows in other cases are suddenly elevated; the face may be drawn to one side, or the facial muscles of both sides may be affected, producing a sudden involuntary grin. The tongue may be affected, and a sudden movement of the tongue as if moistening the lower lip may be so frequently repeated as to produce an inflamed condition of the skin of this area. In one child who had been able voluntarily to produce a movement of the ears, a sudden jerky movement of both ears developed independent of the volition of the patient. Spasm of the muscles of the neck result in a jerking of the head to one side; shrugging of one or both shoulders is not infrequent.

There are usually no other symptoms apart from the motor phenomena. Hysterical outbreaks are occasionally met with in the type of children subject to this affection.

**Diagnosis.**—In rare cases several groups of muscles may be affected at the same time, but they can be easily differentiated from Sydenham's chorea by the purposive character of the movements.

There is a condition of the muscles of the face, most frequently localized to the orbicularis palpebrarum and due to the toxic influence of tea and coffee on the nervous mechanism, which should be distinctly differentiated from habit spasm. The manifestations are lightning-like contractions of the individual fibrillæ, affecting all of these fibrillæ in rapid succession, producing at the most a slight quivering motion of the lids, but never leading to the distinct blinking of habit chorea. This is as frequently met with in adult life as in children, and rapidly yields to treatment when tea and coffee are excluded from the diet. In some of the cases eye strain is a factor.

Habit spasm should also be differentiated from what has been described as impulsive tic (Gilles de la Tourette's disease). Some of the forms of this condition appear to me to be a more serious and widespread affection of the toxic condition, above described, affecting the orbicularis palpebrarum, and due to some intoxication of the system. It is not infrequently fatal. It begins, as a rule, in very early life, although it may occur as late as early adult life. The muscular movements may affect all the voluntary muscles, are lightning-like in character, with marked fibrillary movements. Another group of cases described under this condition presents the same quick action of the muscles, with mental disturbance and the use of foul language. The explosive quick character of the movements, the mental disturbance, and the coprolalia should differentiate it from either Sydenham's or simple habit chorea.

**Treatment.**—In both habit spasm and impulsive tic an underlying cause should be carefully searched for and removed. In the habit spasm about the eyes errors of refraction and loss of muscle balance should be first corrected. The mucous membrane of the nose and the condition of the turbinated bodies should be examined to determine any cause for irritation. The ears and teeth should in the same way receive attention in all cases where the symptoms are referable to any part



of the face. Irritative reflex disturbances in the genitourinary tract, such as phimosis, etc., should be relieved. One case of complex shrugging movements about the shoulders and twisting movements of the trunk resisted treatment until a rough woollen sweater which the boy wore next to the skin had been replaced by proper underclothing. The spasm then rapidly disappeared.

The general nutrition of the body should be brought to a normal standard and a proper discipline infused in a routine way into the child's life. While punitive disciplinary measures sometimes succeed in early imitative cases they frequently do harm. The child should be sent to bed at a definite time early in the evening and compelled to remain in bed an hour after the usual time of rising, both as a disciplinary measure and to secure an added amount of rest for the weakened nervous system. A period of rest in the middle of the day is also advisable. A cold sponge bath or needle bath is valuable as a tonic stimulant if the child reacts well. A simple diet without tea and coffee and with little meat, but with plenty of milk, eggs, and vegetables is indicated. Alterative tonics, such as Fowler's solution, quinine, and strychnine, are sometimes of value; more frequently better results are secured by the use of bromides and other nerve sedatives. The child should be encouraged to inhibit the movements as far as possible.

**Electric Chorea.**—This is a rare disease, first described by Dubini, and is manifested as intensely rapid rhythmic movements in the extremities, rarely in the head and face. The movements may be very violent and have the appearance as if produced by an electric shock. In the severe form described by Dubini as occurring in Italy, paralytic symptoms supervene, and may be associated with epileptiform convulsions. Pain in the head and neck may be present early, and toward the end of the attack atrophy and wasting of the muscles may occur. Fever may be present. The cases terminate in a few weeks or a month from heart failure or coma. A form of electric chorea, probably due to hysteria, has been described by Bergeron. Henoeh has also described a form of electric chorea differing from both the above, and manifested by spasmodic attacks of lightning-like contractions confined to the muscles about the shoulder-blade, it is probably a form of myoclonus.

**Treatment.**—This should be directed to the removal of any underlying intoxication. Free purgation, chloral and the bromides have been used, but to little effect.

**Chronic Progressive Chorea.**—While this disease, described as Huntington's chorea, is typically a disease of adult life, a peculiar condition resembling the adult form developed in a child, the father of a member of a family, all of whom have either died of or at present have Huntington's chorea. About the second year of life choreiform movements resembling the movements of Sydenham's type began in one of the lower extremities and spread to the rest of the body. The movements continued for over a period of two years and disappeared occurred from scarlet fever. Remak and Oppenheim have described



similar condition affecting several children of a woman suffering with permanent hemichorea.

**Diagnosis.**—This disease can be differentiated from Sydenham's chorea, which it closely resembles in the clinical picture, by the presence of the adult form of chorea in one of the parents and the chronic nature of the affection.

**Treatment.**—No method of treatment has produced appreciable results in this disease. Hypnotic suggestion may control the movements temporarily. The mental deterioration demands asylum treatment.

**Organic Choreas. Post-hemiplegic Chorea.**—After some of the cerebral lesions of childhood, which will be later described, a series of movements develops in the palsied arm, to which the name of chorea has been erroneously given. These movements are: (a) gross rhythmic tremors; (b) athetoid movements (slow, snake-like movements of the extremity), which may be constant during the waking hours or brought on by attempts at volitional movement and could not be mistaken for the quick, jerky movements of Sydenham's type or the purposive movements of habit chorea.

Minor athetoid movements may be present, with practically no loss of power in organic lesions sufficiently near the motor fibres of the brain to produce irritation without destruction.

Athetoid movements at times become so annoying that amputation of the offending part has been done, but the results are not in proportion to the gravity of the operation. Recently transplantation of the tendons has been suggested by Spiller with much benefit.

**Prehemiplegic Chorea.**—This is very rarely if ever met with in children, but it is of not infrequent occurrence in adult life immediately preceding an apoplectic attack. It is due to vascular nutritional changes and follows one of the above forms of movement.

## THE CONVULSIVE DISORDERS OF CHILDHOOD.

### REFLEX CONVULSIONS.

The nervous system of the infant is so sensitive to the influence of toxins and reflex irritation that a convulsion is not an infrequent occurrence in the life history of a normal child. It should, however, always be borne in mind that convulsions in infancy or childhood which may be ascribed to slight causes, such as teething, minor infections, etc., denote an unstable condition of the motor nervous system, which may develop a convulsive habit with greater ease and with less cause than in normal nervous system. The onset of the infectious fevers, especially those associated with high fever, are very prone to be ushered in with convulsions. This is probably due more to intoxication than to the irritant disturbance. Peripheral irritation of the gastroenteric tract, intestinal fermentation, intestinal parasites, genitourinary irritation (balanitis), nasopharyngeal irritation (adenoids), delayed dentition, and

rickets are frequent causes of convulsive disturbances, rickets being the most common underlying vice of constitution which predisposes to this instability. Cerebral hemorrhage and other organic lesions of the brain are associated with convulsions. Extreme passive congestion such as that caused by the paroxysms of whooping-cough may also cause convulsions by minute or gross extravasations of blood in the brain cortex.

**Symptomatology.**—The convulsions vary so in the clinical picture that it is rare for two to be exactly alike. They usually come on suddenly without previous warning, and with or without an incidental cry; the body is suddenly thrown in a condition of tetanic spasm, the head is retracted, the eyes turn up, the pupils are dilated, and do not react to light. Clonic convulsions may follow or they may be entirely absent. The mother usually gives a history of "inward spasms," *i.e.*, a purely tonic spasm without the clonic convulsions. In some cases, restlessness and twitching of the muscles of the arms, grinding of the teeth in older children, may precede the convulsive stage. The spasms usually begin in the upper extremities. The body is held rigid, the eyes fixed, the head retracted and breathing is suspended for a short time, as a result of which the face becomes congested. For a minute or two, slight or extensive jerkings of the extremities may follow. After they cease, the child falls into a natural sleep, or more frequently into a state of stupor, or in fatal cases into a deep coma. It is uncommon, except in pure reflex convulsions due to overloading of the stomach, or those ushering in an acute infection, for the convulsions to be single. It is a rule to have repeated convulsions which may be separated from one another by a distinct interval, or the convulsions may follow each other in rapid succession. In such cases, the child may never recover from the comatose condition. It is exceptional to have a fatal outcome of the single isolated convulsions. After the convulsions have disappeared, the child may present nothing abnormal in an examination of the nervous system. It is not infrequent however, to find a weak or parietic condition of one side of the body, which may rapidly disappear. Complete hemiplegia, persistent in type, which later becomes spastic, is not infrequently seen.

The persistence of the convulsive habit is a matter for serious consideration. In unstable children, every effort should be made to prevent recurring attacks and to minimize the danger to the nervous system by lessening the intensity of the individual attacks. In the analysis of 1450 cases of epilepsy, Gowers found that 180 began during the first three years of life. Osler gives a much higher percentage, of 460 cases of epilepsy in children, 187 began during the first three years; 74 of these began during the first year. It does not follow, however, that convulsions during childhood necessarily imply epileptic attacks during later life. Great care, however, should be taken of such children to relieve the developmental period of life from any unnecessary strain on the nervous system, and to guard the child from reflex irritability.

**Prognosis.**—In simple reflex convulsions the prognosis, so far as life is concerned, may be considered to be good; the large mortality in children, as put down in the health reports as due to convulsions, embraces such a large variety of conditions, including the organic palsies of childhood, meningeal hemorrhage, uremia, etc., that they lead to a false impression as to the mortality of this affection. There is, however, not only danger of death from asphyxia, but also a possibility of hemorrhagic extravasation, which may lead to paralysis. Repeated convulsions if not controlled may finally develop into major epilepsy.

**Treatment.**—The same precautions to safeguard the general health of the child and to establish a proper stability of the nervous system, as suggested for epilepsy, should be carried out. Reflex causes should be removed as far as possible. For the treatment of the individual convulsion, the hot bath at 100° to 105° F. in slight cases may be of some benefit; in the severe convulsions it is of little value and takes up time that should be devoted to other measures. Inhalations of chloroform or nitrite of amyl, or equal parts of both, should be continued until the convulsions have disappeared. Enemata to empty the large intestine will be helpful in a great many cases. Small doses of opium in combination with the bromides will usually prevent the return of convulsions. After a day or two the bromides or chloral should be reduced to smaller doses and kept up for at least a week. In acute febrile cases means should be used to reduce the temperature in order to guard against subsequent attacks. Where gastric irritation is present calomel should be used to evacuate the bowels. Convulsions with marked laryngeal symptoms (*laryngismus stridulus*) should be treated on the same principles; the child, however, should be held in an upright position and, if breathing is too markedly interfered with, traction of the tongue or cold douches to the chest should be employed. Rickets, which is present, is often a predisposing factor, and should, of course, receive careful dietetic and hygienic consideration.

### EPILEPSY.

Epileptic attacks in childhood may be divided for purpose of description into (1) Grand Mal, (2) Petit Mal.

**Etiology.**—At least one-fourth of all cases of this disease begin before the tenth year of age, and the great majority of the remainder (at least three-fourths) before the twentieth year. The few remaining cases may occur at any time of life, but a careful investigation of the early history will usually reveal some evidence of epileptic manifestations in childhood. Thus, a young woman of twenty-six years, suffering from epileptic convulsions for the past two years, had attacks of petit mal in childhood, which were not considered of sufficient importance at the time to merit medical attention. Females are more likely to be affected than males. This is especially true about the time of puberty and under the influence of the development of the menstrual period.

Heredity is an especially important etiological factor. The heredity of distinct epilepsy, although present according to some observers in 40 per cent. of the cases, is of not such frequent occurrence as hysteria, insanity, syphilis, and, possibly, alcoholism in the parents. Constant reflex irritation is often a determining factor in the production of epileptic attacks. In an individual with an unstable nervous system, constant reflex irritation along the gastroenteric or the genitourinary tract, if not relieved before the convulsive habit is thoroughly developed, may lead to the formation of a true epilepsy.

**Symptomatology.** 1. *Petit Mal*.—A child with petit mal may show nothing more than a sudden loss of consciousness, lasting from a few seconds to several minutes. The child suddenly stops in its play or in the middle of a conversation, becomes pale, or, perhaps, flushed, has a dazed expression, and either resumes the conversation without any knowledge of its interruption, or may be confused, slightly incoherent, and perform some simple or complex automatic act. In those cases in which the attack is of some duration the child, if he happen to be at the time walking, may continue and suddenly find himself in some strange location. I have known cases of sudden unconsciousness in epilepsy to last as long as an hour, during which time a variety of complex and apparently conscious and rational acts are committed of which there is absolutely no recollection, or only a dim recollection of those at the beginning of the attack. To this form the term *psychic epilepsy* has been applied. Petit mal, or psychic epilepsy, may exist alone or in combination with—

2. *Grand Mal*.—The grand mal, or the epileptic fit, as occurring in childhood, may present any one or all of the following manifestations:

(a) *Aura or signal symptom.* Immediately preceding the attack a warning of its approach is frequently given, which may be of only momentary duration or sufficiently long to enable the patient to protect himself from injury. Most frequently this sensation is a feeling of discomfort or an indefinable sensation beginning in the stomach or some of the other viscera, and either remaining localized there or ascending to the head, when consciousness is lost. In a boy of six years precordial distress and palpitation ushered in the attack. A sensation of a breeze may be present in the extremity. Flashes of light before the eyes, or colored balls may be seen; a peculiar sound or word or sentence may be heard; a peculiar taste or odor or, in some cases, even an idea, a landscape, a vision of creeping bugs, or of snakes may be present before the attack. Slight motor disturbances may be present before consciousness is lost. A few seconds after the beginning of the aura the child falls suddenly and heavily to the floor in a—

(b) *Tonic spasm.* Consciousness is now lost; the head is retracted and may be turned to one side. The extremities and the muscles of the trunk are in rigid spasm, respiration ceases, the face becomes cyanosed, and in a few seconds the—

(c) *Clonic spasm begins.* The child begins to jerk the extremities rhythmically, the arms being slightly flexed and extended. The ex—



tended legs are likewise affected and beat a tattoo with the heels on the floor; the face is involved in the rhythmical contraction, the respirations are stertorous, the eyes are turned upward and may be the seat of jerky movements, the tongue may be bitten and bloody, and frothy saliva may stain the face and clothing. The bladder is very frequently and the rectum occasionally evacuated during the attack. This clonic stage may last from a half to five minutes and then pass over into the—

(d) Comatose stage. The jerking gradually ceases, breathing becomes less stertorous, the muscles become relaxed, consciousness is still lost, and the eyes remain either wide open or half closed. This condition gradually goes over into natural sleep, from which the patient awakens in a semidazed condition with headache and soreness of the muscles and tongue. In some cases automatic actions occur, such as taking off the clothes, running movements, etc. Maniacal outbreaks sometimes follow the attack and a gradual loss of mental power is the rule where the attacks occur at frequent intervals. A monoplegic or hemiplegic paralysis, temporary in character, very rarely follows the fit. The child is always unconscious during an attack of true epilepsy.

**Diagnosis.**—Hysteria, uremia, and simple reflex convulsions may be mistaken for epilepsy. The epileptic convulsion is distinguished from the hysterical convulsion by the loss of consciousness, the sequence of the different stages above described, the rhythmic movements affecting the flexors and the extensors, the relaxation of the vesical and rectal sphincters, and the biting of the tongue. Uremic convulsions may closely simulate the epileptic convulsion, but are easily separated from it by the examination of the urine and the associated vascular symptoms. The reflex convulsions of childhood do not differ from real epilepsy in infancy.

**Prognosis.**—As a general rule, the earlier in life the convulsive epileptic habit becomes established the more incurable it is. In rare cases the convulsions may cease under appropriate treatment as adult life is approached. Where, however, the convulsions occur at frequent intervals, not only is there little hope of control, but distinct mental deterioration may be expected.

**Treatment.**—A careful examination for visceral disturbances and a correction as far as possible is a very necessary preliminary to the treatment of this disease. Reflex disturbances in the nasopharyngeal gastroenteric and genitourinary tracts should be carefully sought for and removed. Particular attention should be paid throughout the treatment to keeping the stomach and bowels in good condition. The bowels should be moved every day; constipation and overloading the stomach are the most frequent determining factors of the individual attacks. Intestinal toxemia due to the improper ingestion of meats is such a deleterious factor that it has been my rule to insist on a vegetable and milk diet. Tea, coffee and tobacco should be absolutely prohibited. A life as free from excitement as possible, preferably in the country, should be enjoined. Regularity in habits of eating, sleeping, and exercise is

necessary in order to restore as far as possible a proper balance and regularity of function of the nerve tissues. The exercise should be carefully regulated to secure a proper condition of the muscles, with the least excitement and the least fatigue. A period of sleep in the middle of the day lessens the mental and physical fatigue and prevents the early night-sleep from being too intense. A large number of cases have their fits at night, and usually when sleep is deepest.

Many drugs are recommended for the cure or control of this affection. The bromides are by far the best medicinal agents at our command. Sufficient bromide should be given to control the attacks. In nocturnal epilepsy as high as 3.90 gm. (1 dr.) of sodium bromide may be given in a single dose before the child retires. A much smaller quantity may be sufficient, but this is a matter of experiment in each individual case.

When the attacks occur during the day from 0.324 to 1.3 gm. (5 to 20 gr.) doses may be given after each meal. When the attacks occur at periodic intervals (approximately every month in a case at present under my care) the dose may be doubled a few days previous to the time of the expected attack. Where control of the convulsions is secured, both the hygienic and drug treatment should be continued for at least two years after the last attack. Arsenic in the form of Fowler's solution assists in controlling skin manifestations of the bromides. Acetanilid, phenacetin, chlorotone, may be combined in 0.324 gm. (5 gr.) doses with the bromides, or given separately when it is considered advisable to intermit the bromide treatment, on account of gastric disturbance or mental symptoms. Care, however, should be used to prevent cardiac depression from the use of these drugs.

*Solanum caribolensis* in 1.25 to 3.75 c.c. (gtt. xx to f5j) of the fluid extract may also be used as a substitute for the bromides. The treatment of the individual attack consists merely in prevention of injury to the tongue or the person. A towel inserted between the teeth and a pillow placed beneath the head meet these requirements. When one convulsion follows the other in rapid succession (epileptic status) free purgation should be obtained. Chloral and bromide by rectum are most satisfactory as sedatives. The patient may be bled, but very young children do not bear the loss of much blood.

### HYSTERIA.

Hysteria is a comparatively rare disease of childhood. It is the typical functional nervous disease for which there is no known pathology.

**Etiology.**—It is much more frequent in the female sex, especially as adult life is approached. It is also of much more frequent occurrence in the Latin races. A neurotic heredity is very frequently present. The "germ" of hysteria may be said to be present in every female child; and in those of a nervous temperament, the worry of forced schoolwork, overwork, fright, or any intense emotional disturbance may determine

an outbreak. Masturbation is an important etiological factor in both sexes and must not be overlooked in girls.

**Symptomatology.**—The clinical picture of hysteria varies so widely that it would be impossible in a short space to give an adequate account of its protean manifestations. We may divide the symptoms into several groups, but it must be remembered that every possible combination of these may occur. The most important and diagnostic group of symptoms may be considered to be:

1. *The Sensory Manifestations.*—The pain in children is apparently of a very agonizing character and may be referred to any part of the body. It is usually associated with hyperesthesia so intense over the part affected that the slightest touch of cotton will cause the patient to cry out. The hyperesthesia, while most intense over the seat of pain, may be present to a lesser degree over one-half of the body, or may be localized in regular or fantastic forms to an extremity or portions of the trunk. Like the anesthetic disturbances, it does not correspond to the anatomical distribution of the nerve supplying the part, and this fact, together with its exaggeration as compared with the tenderness of inflammatory or other organic conditions will usually stamp its functional character. Anesthesia is of much more frequent occurrence and follows the same rules. In rare cases there may be universal anesthesia of the skin and the superficial mucous membranes, the cornea, however, being practically never involved. A pin-prick over the anesthetic areas is usually not followed by bleeding. In rare cases there may be other vasomotor disturbances, such as local or extensive edema. In one case a circumscribed firm edema of the popliteal space associated with a band of hyperesthesia about the knee and anesthesia of the leg below the knee was mistaken for a tumor formation.

2. *Motor Manifestations.*—These are usually associated with either of the sensory disturbances above described in the part affected. Hysterical paralysis may affect an individual group of muscles, as the muscles of the larynx and produce aphonia; more commonly an entire extremity may be affected, or that they may be a hemiplegic type. The face, as a rule, is not involved. Very rarely a quadriplegia may be present. The reflexes are always present, although it may be necessary to distract the attention of the child before they can be elicited. As a rule, the reflexes are quick. Although anesthesia may be present, pain may also be complained of, and tenderness to pressure may be present over the muscles or nerves. The presence of the reflexes, the history of the onset, and the influence of suggestion will separate this paralysis from that due to neuritis or organic disease of the brain. Hysterical contracture or hysterical spasm may likewise affect a group of muscles, an entire extremity, or several extremities. It is also associated with the sensory disturbances, and likewise yields to suggestion. Hysterical tremors or gross jerkings may affect a single member or be widespread over the entire body. They are usually rhythmical in character, although they may assume any form, but do not, as a rule, follow the type of any of the organic affections. A combination of contracture of some muscles



associated with relaxation of others in the abdominal region produces the false or phantom tumors. These disappear under ether.

The hysterical convulsion usually affects all the voluntary muscles at one time or other. While the French clinicians have described a regular series of psychic and motor events during the course of the convulsion, these are more the result of suggestion than of any innate tendency to follow a definite clinical picture. The convulsions may follow any form; they may be brought on by excitement or emotion or occur spontaneously, but usually in the presence of persons from whom sympathy may be expected. A sudden tetanic spasm, during which the body is highly arched, the patient perfectly conscious or apparently unconscious, but receiving impressions from without, with respirations normal or jerky in character, may be the only manifestation. This, however, is usually followed by wild cries, irregular jerkings of the arms or legs, or at times clawing or swimming movements. After the attack passes off the patient remains in an excited state, but does not fall into the sound sleep of general epilepsy. Patients stationed in an epileptic ward of a general hospital, where they may observe real epileptic fits, present in their own convulsions a picture that can be easily distinguished from true epilepsy. There is, as a rule, no real loss of consciousness, and the jerkings do not follow the flexor and extensor type. The tongue is never bitten nor is the bladder evacuated during the hysterical convulsion, with a possible exception of those cases where the repeated questioning of the examiner along these lines suggest to the hysterical patient the importance of such facts in diagnosis. The pupils are normal during the hysterical convulsion. It must be remembered, however, in this connection that hysterical outbreaks may follow a true epileptic seizure.

3. *Visceral Symptoms.*—Inability to swallow, due to an hysterical spasm of the esophagus, can be easily differentiated from true stricture by the passage of a full-size bougie. Hysterical hiccough may occur alone or be associated with aphonia or cyanosis. The swallowing of air associated with either tremendous distention of the abdomen or prolonged belching attacks occasionally occurs, and may be associated with hysterical convulsions. Hysterical cough with hemoptysis, anorexia, and loss of weight has been mistaken for pulmonary tuberculosis.

Hysterical anorexia and hysterical vomiting may lead to marked emaciation. Hysterical diarrhea is not infrequent. Bradycardia or more frequently tachycardia may be associated with intense precordial pain.

4. *Mental Symptoms.*—In combination with any of the above groups of symptoms an emotional atmosphere surrounds the patient which is very characteristic. Craving for sympathy is rarely a verbal request of symptoms such as met with in neurasthenia, but rather a demand by action such as causeless crying attacks, expression of intense pain, anxiety or fear, or some of the above motor manifestations at an opportune moment. A nervous irritability associated with laughing or crying spells may become so marked as to lead to distinct mental alienation. The



hysterical insanity is merely an accentuation of intense emotion and excitement, and may be either very active or be associated with such depression as to lead to simulated or real attempts at suicide.

**Diagnosis.**—The main points of a diagnosis have been considered under the individual symptoms. Hysteria should never be diagnosed until organic disease has been entirely excluded or the organic element dissociated from the functional manifestations. The previous history of the case and the influence of suggestion in controlling individual symptoms are the most important factors in making the diagnosis. While the hysterical manifestations closely resemble organic disease there is always something atypical, and the exaggeration alone is usually sufficient to call the attention to the possibility of hysteria, which may be confirmed by the sensory manifestations. In childhood more than at any other time of life do we meet with monosymptomatic hysteria. In rare cases it may be even necessary to hypnotize the patient in order to dispel a paralysis, a tremor, or contracture.

**Treatment.**—To protect children of a nervous temperament from the development of hysteria and allied functional disorders, great care should be used in the education of the child. This refers as much to home training as to school education. A firm discipline tempered with kindness is very necessary in both places. Regular methods of life, with plenty of out-of-door exercise; a good, nutritious diet, with little meat and no tea or coffee, should be insisted upon. Care should be used, especially in growing girls, to prevent overwork at school and to relieve the child as far as possible from the worry of examinations. When hysteria develops it may be necessary to treat both the individual attacks and the disease itself. In the milder cases a change of living atmosphere of the patient, under the guidance of a trained nurse or a companion at some country resort away from the influence and sympathy of over-anxious relatives and friends, is all that is necessary. In all cases underlying organic or functional disturbances of the viscera should be carefully sought for and eliminated. Constipation should be relieved by appropriate remedies, and proper sleep secured by the use of bromides, trional, etc. In all but the most severe cases I have found a modified rest treatment either at home, or, better, at some health resort, the most beneficial method of treatment. The regulations once established should be absolutely insisted upon. A physician should see the patient every day or every second day, and carefully inquire into the details of the treatment. Directions as far as possible should be written out in detail. Where directions are simply given and no further inquiry made it may be safe to assume in nearly every case that violations will frequently occur.

Apart from the beneficial results to the exhausted and unstable nervous system, the discipline and moral encouragement by the physician are of value in strengthening a weakened will-power. Twelve hours rest at night should be insisted upon. It is quite immaterial whether the patient sleeps all this time or not. At least two hours' rest in bed in the middle of the day, at a definite prescribed hour, is necessary to

overcome the fatigue developed during the day and to give the nervous system a chance to recuperate. The exercise should be carefully regulated and selected in such a way as to give as much pleasure as possible with the least excitement. In the severe cases, and in those in which the muscles are soft and flabby, well-regulated massage should precede the out-of-door exercises. Electricity is also of value both as a stimulant tonic and to secure a proper condition of the muscles. All of the regulations should be so arranged at fixed intervals as to keep the patient occupied and to keep the mind as far as possible away from the local symptoms and the patient herself. For the individual symptoms, a suggestion that there is a constant improvement will usually be all that is necessary. If the patient's mind is not too much concentrated on any individual symptom, it is much better to disregard treatment in that direction until the systematic treatment is thoroughly developed and the confidence of the patient secured. A firm, hopeful, confident attitude, with a proper tactful dispensation of sympathy or harshness, are necessary qualifications for the physician to secure results in the handling of these cases. The relatives and friends of the patient should either not be permitted to see the patient at all or only at intervals, and then as a reward for the control of some particular manifestation of the disease. Overfeeding may be necessary in cases of low nutrition, and a very careful discrimination in the use of massage, exercise, and dieting in flabby, fat individuals. Sedative tonics in conditions of excessive nerve irritability are often indicated. I have found the following prescriptions of considerable value:

R—Sodii bromid.	0.08 to 1.3 gm.	(gr. v to xx).
Tr. nucis vom.	0.03 to 1.0 c.c.	(gtt. v to xvj).
Tr. cinchona comp.	2.00 c.c.	(3ss).
Aq. dest.	ad 15.00 c.c.	(3ss).
M. et sig. 4 c.c. (5j) t. i. d.		
R—Sodii bromid.	0.08 to 1.3 gm.	(gr. v to xx).
Eltx. valerianæ ammoniac	2 to 4 c.c.	(3ss to 3j).
M. et sig. 4 c.c. (5j) t. i. d.		

Valerian, asafetida, paraldehyde, and other nauseous drugs are frequently used, but are mainly of value on account of their disagreeable taste. In anemic conditions the iron preparations are serviceable.

In very severe obstinate cases the full rest treatment as outlined by Weir Mitchell gives the best results. The patient should never be treated at home, but removed to a hospital or some institution where absolute seclusion in a quiet room can be secured. An intelligent nurse familiar with the treatment, or, better, a nurse especially trained for it and congenial to the patient, is essential. Absolute rest in bed with no even permission to read, write, feed, or otherwise care for herself should be insisted upon. Massage, electricity, and overfeeding are other essentials of the treatment. The same rules as to regularity, system, and control as above outlined in the modified rest treatment should be carried out. It may be necessary in some cases to start on the simple milk diet and gradually add other food as soon as the system is educated to take care of it. Exclusion of visitors and even of news of the outside

world is at first necessary, and later permitted according to the rules above laid down in the modified treatment. The greatest care must be used toward the end of the treatment in restoring the patient to normal mental and physical surroundings. The patient should at first be permitted to sit up for a short time, and this time increased if no untoward symptoms are produced. Fatigue, nervousness, and insomnia are indications that too much is being attempted. The same is true when the patient begins to walk and to take out-of-door exercises. Responsibility of gradually thinking for herself and of deciding as to other responsibilities of life should be gradually shifted from the physician to the patient until a normal condition obtains. A regular method of life should be insisted upon for a long time. The education of a proper mode of living and of the care of the nervous system are not the least of the benefits to be expected from this treatment.

While hysteria is prone to recur in those who have once thoroughly developed the disease, normal nervous health rests to a great extent in the hands of the patient, and if she has profited by the lessons learned, and the influence of rest, regularity, and system in keeping the nervous system at its highest point of efficiency, thus avoiding any unnecessary strain, there is little likelihood in the majority of cases of a return of the affection.

#### NEURASTHENIA.

While Neurasthenia is an uncommon condition in childhood, mild and, rarely, more severe forms are occasionally met with. It usually develops in children of a high-strung nervous temperament who are being pushed too fast at school, associated with the worry of an oncoming examination or possibly of some home affliction. It may follow an influenza. Masturbation in growing children may be an important factor. Eye strain is frequently present. It has also been observed in infancy where a baby has been kept agitated and disturbed.

**Symptomatology.**—Mental and physical fatigue are the predominating symptoms. The child becomes moody, introspective, and in a child approaching adult life suicidal tendencies may be manifested. Obsessions with impulses to do a wrong thing or to satisfy a morbid desire may be associated in the severe forms with loss of memory, failure to concentrate the attention, and intense excitability or depression. Pain in the head or back may be complained of, but there are no objective disturbances of sensation. A subjective sensation of cold water running over the body or ants crawling over the skin may be present. While the reflexes are usually quick, there are no paralyses or other motor manifestations other than fatigue and a fine tremor after excitement or mental and physical exertion.

**Diagnosis.**—An incipient tuberculosis, an unsuspected cardiac or renal disease is so frequently mistaken for neurasthenia that we are only justified in making this diagnosis after the most careful examination and exclusion of organic disease.



**Treatment.**—The care of the child, the hygiene of its life, and the treatment of the disease do not differ from that above outlined for hysteria. An infant should be allowed to lead a perfectly normal life without disturbance by relatives and friends.

#### THOMSEN'S DISEASE.

This may be considered to be an hereditary disease affecting several members of the same family and is called Myotonia Congenita. In Thomsen's family the affection could be traced through five generations. In some cases the heredity is missing, and isolated individual members of a family may be affected. Sporadic cases presenting the same clinical picture are occasionally met with. Transitory conditions resembling this disease also occur. It is a rare condition, but I have seen three cases in European clinics, and one case which came under my own observation.

**Pathology.**—The nervous system so far as has been studied has shown no pathological lesions. Hypertrophy of the primitive muscle fibres with multiplication of the muscle nuclei has been found.

**Symptomatology.**—The disease develops early in childhood and is manifested by a rigidity of the muscles when a voluntary movement is attempted. If the child be in a sitting posture and attempts to get up and walk, the muscles of the leg and back become rigid, and it is only after repeated attempts at motion that sufficient relaxation occurs to permit free movement. With each successive movement more freedom is gained, until after several steps a normal condition obtains. The same condition is present in the arms and rarely in the face and laryngeal muscles. Exposure to cold and emotional excitement accentuate the symptoms. Mental weakness has been noticed. The muscles are normal or overdeveloped, but are usually weak in comparison to the volume of muscle tissue. As adult life is approached a pseudo-hypertrophic condition with deposits of fat between the muscle fibres and marked motor weakness may develop. A tap on the muscles produces a local spasm, which lasts several minutes before relaxation occurs. The same is true of the reaction of both muscle and nerve to electric currents. There is no known treatment that has much influence on the course of the disease. Spontaneous arrest has been noted.

Eulenberg has given the term *congenital paramyotonia* to a modification of the above disease. It is also hereditary in character, of a family nature, and manifested by a tonic spasm lasting from a few minutes to several hours, brought on by exposure to cold. There is an absence of increased mechanical excitability and also of the myotonic electric reaction.



**PARAMYOCLONUS MULTIPLEX.**

This is also an hereditary affection and is one of the rarest of the motor diseases, although not so rare as Thomsen's disease. Males are more frequently affected. Intense fright, straining, or other emotional disturbance has been blamed for it. Rapid rhythmical contractions, varying from fifty to one hundred and fifty to the minute, affecting individual muscles or groups of muscles and, as a rule, symmetrical muscles, are the chief clinical manifestations. Tremor of the muscles may be present during the intervals between the grosser clonic movements. The face muscles are usually exempt and in this respect it is unlike chorea or the other clonic spasms. The muscle contractions are so rapid and of such short duration that the movements in the extremities produced are, as a rule, not marked. There is no change in the electric excitability. It is more frequent in adult life, although it may occur in children. If associated with epilepsy it is termed myoclonus epilepsy. It is differentiated from Sydenham's or electric chorea by the rapidity of the contractions, the absence of the irregular movements of the extremities, and the course of the disease. A very few cases have been entirely cured. The prognosis in most cases is serious.

**Treatment.**—Alterative tonics, nerve sedatives, increase of the body nutrition, and regulated gymnastic exercises sometimes do good.

**NOCTURNAL ENURESIS IN NERVOUS DISORDERS OF CHILDHOOD.**

Children otherwise of good habit during the day not infrequently manifest disturbances of micturition during the night. This usually occurs during the soundest sleep and this in itself may be sufficient in some cases to account for it. In other cases it is due to faulty training and an aversion on the part of the child to permit an interruption of its sleep. In rare cases, and this is especially true of those in which night terrors are associated, it may be a manifestation of an oncoming or developed epilepsy. In the latter cases the tongue may be bitten, or headache or drowsiness be present during the following day. While night terrors may be merely the manifestation of the fear engendered upon waking from a sound sleep by a frightful dream it should be borne in mind that this condition is most frequently present in children of a nervous temperament and of neurotic heredity. It occurs usually between the third and eighth year of life and may persist even to adult life. The history of night terrors in children occurs so frequently in the history of epileptic children, and in those who have been cured of the convulsive habit, that the relation of enuresis and night terrors to epilepsy should always be borne in mind. Other factors are mentioned in the section relating to the Diseases of the Genitourinary System.

**Treatment.**—Although at times a very stubborn condition it will, in children of normal mentality, yield to careful training. Salt and salty

foods should be carefully eliminated from the diet, and liquids excluded after the middle of the day. The time of the occurrence of the micturition should be carefully noted and the child awakened from half an hour to an hour before this time and the bladder evacuated. Where more than one evacuation of the bladder occurs during the night, the child should be awakened at frequent intervals. If drugs be used the tincture of belladonna should be given in ascending doses until physiological results are obtained. It is useless in small doses or where given alone without the assistance of the above directions. When night terrors are present the bromides are indicated. The hygienic regimen outlined under Epilepsy should be carried out if there is even suspicion of a beginning epilepsy. Corporal punishment frequently produces results opposite to that desired. The treatment of incontinence of urine due to myelitis, Pott's disease, spinal tumor, encephalitis, etc., is given under these diseases. In retarded mental development, idiocy, etc., treatment is practically useless.

## CHAPTER XXXVI.

### ORGANIC NERVOUS DISEASES—DISEASES OF THE NERVES AND SPINAL CORD—ABIOTROPHIC DISEASES.

#### ORGANIC NERVOUS DISEASES.

##### DISEASES OF THE PERIPHERAL NERVES.

INFLAMMATION of the nerves, Neuritis, may be localized to a single nerve, it may affect several nerves, or it may involve almost if not all of the peripheral nervous system. To the two latter forms is given the term *multiple neuritis*.

**Etiology.**—While idiopathic forms of neuritis have been described and exposure to cold and wet given as a cause, it may be said, as a general rule, that if traumatism or pressure on the nerves be excluded it may be assumed that the neuritis is caused by an underlying intoxication or the presence of some micro-organism in the nerve.

**Pathology.**—From a pathological standpoint two distinct forms of neuritis may be recognized—a *parenchymatous* and an *interstitial*. *Parenchymatous neuritis* is a toxic degeneration affecting the axis cylinder and its myelin protecting sheath, and with no changes or very minor changes in the connective tissue. This condition, the best type of which is seen in lead palsy, lacks the characteristic manifestations of an inflammatory process and is more strictly a degeneration than an inflammation. There is no elevation of temperature, no redness of the nerve, and the bloodvessels of the interstitial tissue are neither surrounded by or infiltrated by hemorrhages or small cells. In a well-developed case the myelin becomes swollen and degenerates into small globules of fat, the axis cylinder becomes granular, and is finally broken down. In progressive cases these materials are absorbed and nothing may remain but a connective-tissue band; in other cases the process may stop at any one of the above stages, followed by, first, regeneration of the axis cylinder, and later of the myelin sheath from the nuclei of the connective-tissue sheath of Schwann. This condition of the nerve is the same as that seen after cutting off the blood supply, after prolonged pressure, or destruction of the cells of the anterior horn of the spinal cord. We most frequently meet with this form of degeneration in the chronic intoxication of lead, and in certain infectious processes such as tuberculosis, diphtheria, etc.

In true inflammation of the nerves, *interstitial neuritis*, the nerve is swollen, congested, of a thicker and redder color, as a rule, although in advanced stages it may be yellow from the presence of pus and

serum. On microscopic examination, besides the changes above noted in parenchymatous degeneration, there is a marked congestion of the bloodvessels, capillary or diffused hemorrhage, and an outwandering of leukocytes, all of which exert a toxic and pressure influence on the nerve fibres. Above and below the point of active inflammation degeneration of the nerve fibres in a distal direction from its nutritive cell connection may be seen. Here again the process may go on to complete degeneration, or it may stop with complete or partial regeneration at any stage of the process. The process of regeneration is, as a rule, very slow, varying from six weeks in the milder types to as many months in the more severe forms. Injuries to nerves may set up an inflammatory process, or an injury to the nerve fibres may produce a secondary degeneration without evidence of inflammatory degeneration, giving a picture similar to that of parenchymatous degeneration. This process may be slight or advance to complete degeneration. The latter occurs where scar tissue develops in such a way as to completely interfere with the transmission of impulses or the regeneration of the axis cylinder. The same is true of the effects of tumors of nerves or the results of the inclusion of a nerve in the callus from a fracture.

**Symptomatology.** 1. *Parenchymatous Neuritis.*—The type of this form of neuritis is that seen in diphtheria. There is no fever, pain, or tenderness along the course of the nerve. The only symptom present is a paralysis in the distribution of the nerve affected, which is usually complete and may be associated with loss of sensation in the skin area supplied. Trophic influence may also be affected, and wasting may occur. In the milder forms the sensation is not disturbed. In diphtheria the nerves to the palate, the extraocular muscles, and those of the lower extremity are usually affected. Prognosis, except in those cases where the vagus is affected, is good, recovery usually taking place in from six weeks to six months. In lead poisoning the musculospiral nerve is usually affected on both sides, with a resulting bilateral wrist-drop.

2. *Interstitial Neuritis.*—The toxins of the infectious fevers (influenza, typhoid, malaria, bubonic plague, pyemia, syphilis, leprosy, beriberi, alcohol, arsenic, mercury, zinc, ether, bisulphite of carbon, and cachectic states, such as cancer, are the most common causes. The symptoms are those of any acute inflammation. If the neuritis be sufficiently extensive there may be slight fever. When a single nerve is affected the symptoms are of course localized to the distribution of this nerve. Pain is the predominating symptom. This may be of a dull or of an intense stabbing character. Tenderness is marked along the course of the nerve and not infrequently in the palsied muscles. In superficial nerves the nerve may be distinctly swollen to palpation. The function of the nerve (motion, sensation, nutrition) is partially or completely destroyed. In the early stages tingling and formication may be associated with slight hyperesthesia; this rapidly gives way to loss of sensation and motor paralysis. The reflexes in the distribution of the nerve affected are lost. I have never seen a case where I felt justified in



making the diagnosis of neuritis with the reflexes normal or increased in the distribution of the affected nerve. Trophic disturbances—wasting of the muscles, glossiness of the skin, local edema, defective or perverted nutrition of the nails—develop in some cases early, in other cases late or not at all. The trophic disturbance is early manifested by the reaction of the muscles to the electric current. There is complete failure of reaction to the rapidly interrupted current, and the reaction to the galvanic current is slow and vermicular instead of a normal quick contraction. This slow contraction is a more positive and diagnostic sign than the change in the formula. Instead of the stronger contraction being obtained when the cathode is applied to the affected muscle and closed, the stronger contraction is obtained when the anode is applied. When multiple nerves are affected the term *multiple neuritis* is employed. While alcohol is the most common form of multiple neuritis met with in the adult that due to the infectious fevers, or the metallic poisons is most common in childhood. In influenza all four extremities may be involved and the symptoms be associated with considerable fever. A fatal termination in such cases may ensue from involvement of the cardiac nerves.

**Diagnosis.**—The absence of the reflexes, the associated motor and sensory paralysis, the change in the electric reaction, and the distribution of the symptoms corresponding to the anatomical distribution of the nerves will usually make the diagnosis. In exceptionally rare cases the inflammation may extend to the spinal cord, with the production of myelitis. Disease of the spinal cord may be excluded by the absence of the involvement of the bladder and rectum, and of marked sensory changes on the trunk. In multiple neuritis the tenderness over the nerves with the preservation of the bladder and rectal function will differentiate this disease from myelitis.

**Prognosis.**—The prognosis in any given case of neuritis must depend on a careful study of the local manifestations for some time. Where the loss of nerve function is complete and the reactions of degeneration develop early, and where there is a marked tendency of a progressive type for the muscles affected to fail to react to increased quantities of the galvanic current, and where other trophic manifestations develop early, the prognosis is grave for return of function. If it return at all it will only be after many months of careful and painstaking treatment. Where the electric reactions are only slightly disturbed or develop some time (weeks) after the onset the prognosis is favorable. In those cases where no change of the electric reactions are noted return of function may be expected in several weeks.

**Treatment.**—Rest of the body and absolute rest of the part affected are absolutely necessary. Any underlying intoxication or pathological lesion causing pressure should be removed as early as possible. The general body functions, and especially the gastroenteric tract, should be brought into a normal condition as soon as possible in order to prevent any added intoxication. To relieve the intense pain phenacetin in doses of 0.06 to 0.3 gm. (1 to 5 gr.) or combined with salicylates is valuable. It

may be necessary in some cases to use morphine. A single blister or multiple blisters along the course of the nerve or the application of the actual caustery both gives relief and has a beneficial influence on the inflammatory process. As soon as the acute inflammatory symptoms have subsided gentle massage and hypodermic injections of strychnine, 0.00012 to 0.0005 gm. ( $\frac{1}{80}$  to  $\frac{1}{16}$  gr.), into the affected muscles should be used. The galvanic current is also of value in securing restoration of function.

#### SPECIAL FORMS OF NEURITIS.

**Obstetrical Palsies.**—These are most often brachial birth palsies and are due to traction, secondary to the manipulations necessary in delivery. In all these cases in which severe traction upon an arm or stretching of head to one side (Clark, Taylor and Prout) is necessary, the brachial plexus is torn and lacerated at Erb's point, the junction of the fifth and sixth cervical nerves. In rare cases the paralysis may be bilateral. The degree of paralysis depends upon the extent and intensity of the injury. The entire arm may be completely paralyzed, or more frequently the upper arm is paralyzed with a fair amount of function retained in the forearm and hand. This form of paralysis is most frequently mistaken for paralysis due to brain lesions. The paralysis of the cerebral palsies is spastic in type, whereas this type of paralysis is flaccid, with lost reflexes and reactions of regeneration.

The prognosis depends upon the same rules as given above in cases of neuritis.

**Treatment.**—Treatment is by massage and electricity, carried out as soon as is practicable. If after a year the paralysis is persistent resection of the nerves may be required (p. 890).

A peripheral paralysis may be prenatal in type, and possibly due to a malposition of the fetus in utero. The paralysis in a case of Dr. Burk's was present at birth and associated with clubhands and clubfeet. The reflexes were present in the forearms, but were absent in the upper arms. This was probably a case of plexus palsy, due to a malposition of the fetus in which pressure was exerted on the brachial plexus on both sides.

Facial paralysis has been produced by the pressure of the forceps on the facial nerve (*vide infra*).

**Facial Palsy (Bell's Palsy).**—Paralysis due to lesions of the seventh nerve may develop at any time of childhood and are due to the same causes as in adult life. The most frequent of these is exposure and is commonly termed rheumatic palsy. This form of the disease is probably due to some infection. Two of the eighteen cases which came under my observation during the past year were children. It usually follows exposure to a draught, although no such history may be present, and outside of a pharyngitis or tonsillitis no evidence of rheumatism is usually present. The next most frequent cause is disease of the middle

ear. This may be simply an involvement of the nerve by a purulent process, or, more frequently, it follows operation on the middle ear with traumatism to the nerve. The third and least frequent cause is the involvement of the seventh nerve within the skull by meningitis, fracture at the base, inflammations, tumors or abscess of the pons between the nucleus of the nerve and its exit.

**Symptomatology.**—Inasmuch as the seventh nerve is purely a motor nerve to the muscles of the face, the symptoms are merely a more or less complete paralysis of motion, with secondary wasting on one side of the face. The child is unable to close the eye, every attempt being associated with an upward movement of the eyeball (Bell's symptom). The forehead is flat on the affected side, and there is an absence of wrinkling when the brows are elevated. The mouth droops and there

FIG. 176



Facial paralysis. (Bell's paralysis.)

is an absence or flattening of the nasolabial fold. There may be a spot of tenderness at the exit of the nerve from the skull. In rare cases pain may be complained of, but this will usually be found to be due to an involvement of the fifth nerve, and sensitive areas will be present at the exit points of this nerve. In severe cases degeneration of the muscles will occur, and where recovery of function does not take place a secondary contraction of the affected muscles may pull the face toward the affected side in such a way as to give the appearance as if the opposite side were paralyzed. In mild cases the symptoms will be more plainly brought out by forcible closure of the eyes or by getting the child to laugh, pout, or show the teeth. (See Fig. 176.)

**Diagnosis.**—From lesions in the brain above the nucleus Bell's palsy can easily be differentiated by the absence of any symptoms other than

those referable to the face. In cerebral lesions a paralysis of an arm or leg, sensory disturbances, and other cranial nerve lesions will be present.

**Prognosis.**—In cases secondary to operative attacks on the ear or the mastoid the paralysis is usually complete and permanent. The majority of cases of the rheumatic class get well after a longer or shorter period. Prognosis can be fairly accurately made by a study of the electric reactions. If the muscles react with a faradic current at the end of a week, and if the reaction to the galvanic current is quick without change of the formula, complete recovery within six weeks may be expected. If, on the other hand, faradic irritability be lost and the reaction to the galvanic current be slow, but where the cathodal-closing contraction is still greater or at least equal to the anodal-closing contraction, recovery need not be expected in less than six weeks and will probably take between three and six months. In those cases where the contraction is very slow and where the anodal-closing contraction is greater than the cathodal-closing a few days after the onset, and where larger quantities of the galvanic current are necessary to produce a contraction from day to day, an unfavorable prognosis should be given, and if function returns at all it will be only after one or two years of careful and painstaking treatment.

In some cases the superior distribution regains its function first, but in the larger number function returns first in the lower distribution and later, if at all, in the superior distribution.

**Treatment.**—The milder cases recover rapidly without any special treatment. In the severer cases a blister or other form of counterirritation midway between the angle of the jaw and the mastoid process should be applied. Electricity should not be begun for at least a week after the onset. A mild galvanic current sufficient to secure a mild contraction of the affected muscles should be used. The smallest possible current to secure contractions should be employed, and should never be so strong as to produce pain or vertigo. It is always a safe rule in using electricity about the head to apply the current after an increase to one's own mastoids, and note the effect before applying it to the patient.

Medicinal agents produce little result. If there be an associated involvement of the fifth nerve, or even without this if there be redness of the throat, salicylate of soda may be used. Alterative tonics such as the tincture of *nux vomica*, Fowler's solution, or the iron preparations may be given.

In cases where the degeneration has been progressive and where there is no evidence of restitution of function either to volitional effort or to the electric stimulus, nerve transplantation has been tried with fair results. (Taylor, Clark.) The facial is cut and its distal end is inserted by lateral anastomosis into the sheath of the hypoglossal nerve. Frazier states that, as a rule, the sooner the operation is done the better the results to be expected. When we believe the nerve to be destroyed, as after operations on the middle ear, etc., operation should be performed without delay. If in doubtful cases at the expiration of six months there is not the slightest sign of



recovery, operate at once. As to operation in cases of long standing (i. e., two up to twenty years) each case must be judged from the standpoint of the electric excitability of the facial muscles. If the facial muscles are completely atrophied and will no longer respond to galvanic stimulation, the prospects of restoration of function are extremely doubtful. For a full discussion of this subject with a discussion of the technique of the operation, etc., see Frazier, *Pennsylvania Medical Journal*, June, 1904, vol. vii., No. 9.<sup>1</sup>

**Root Palsies.**—Forcible stretching of the extremities, especially of the arms in gymnastic feats, may give rise to a degenerative condition which has been ascribed to lesions of the nerve roots. The symptoms do not differ from that of the obstetrical palsies or other forms of plexus neuritis except in the persistence of the symptoms. When the eighth cervical and first dorsal roots are affected there may be dilatation of the pupil, with unilateral sweating of the face on the affected side (Klumpke's paralysis). The treatment is the same as that given above under Neuritis. The *prognosis* is unfavorable, when the roots are involved. Operative procedures on the nerve trunks give better results.

## DISEASES OF THE SPINAL CORD.

### ACUTE ANTERIOR POLIOMYELITIS. SPINAL PARALYSIS OF CHILDHOOD.

The peculiar blood supply of the spinal cord whereby the gray matter of the anterior horns receives its nutrition almost direct from the anterior spinal artery, exposes this portion of the spinal cord to a more direct attack from infectious or toxic material in the circulating blood than occurs in the other columns of the cord. It was at one time thought that an inflammatory process circumscribed and localized to a limited area was always localized to the anterior horns, but recent observations have shown a similar process affecting the posterior spinal ganglia in herpes zoster.

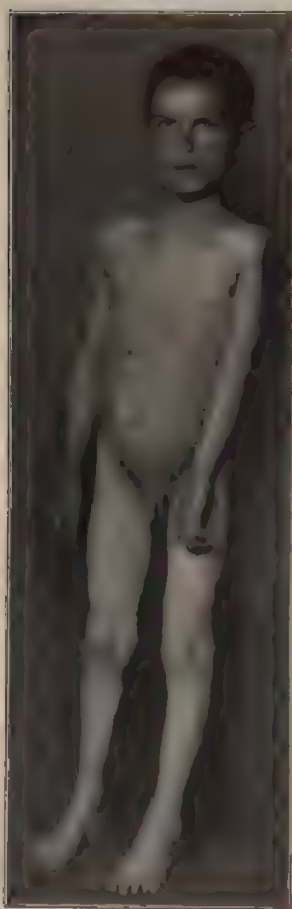
**Etiology.**—Several epidemics have been described. The disease is probably of an infectious nature, and although isolated organisms have been found in the cerebrospinal fluid no distinct causative agent has been isolated. While as a rule children in robust health are affected, it is not infrequent in a large number of cases to get occasionally a direct antecedent history of scarlet fever, measles, or gastroenteric disturbances. Mothers usually blame some slight injury, but traumatism as a factor need not be considered.

**Pathology.**—The lumbar region of the cord is most frequently affected, and next to this the cervical region. The pathological process varies from a simple acute congestion to an active acute inflammation. In the

<sup>1</sup> See also Clark, Taylor and Prout, *American Journal of the Medical Sciences*, October, 1905, for a full report on this class of cases.

later stages there is a dilatation of the arteries, with small capillary hemorrhages and a heaping up of small, round cells about the vessels. Immediately surrounding the central focal area, where the nerve cells are undergoing complete degeneration, there is a perifocal zone of congestion in which the nerve cells, although affected, are not beyond the

FIG. 177



Poliomyelitis affecting the left arm and right leg.

stage of regeneration, and in which zone the axis cylinders are swollen. This zone may extend to the white substance of the cord in the immediate neighborhood of the anterior horns. In cases that have existed for a long time, circumscribed atrophy occurs, with decrease in size of the anterior horns and a partial or complete absence of the functioning ganglion cells. The pathological process may be found as high as the medulla in rare cases, and an analogous, localized, circumscribed inflammatory process may be found in the cerebrum (encephalitis).

**Symptomatology.**—A perfectly healthy child or one convalescing from scarlet fever or other acute infection may awaken in the morning with a paralysis of a group of muscles or an entire extremity. More frequently the disease may come on with fever, ranging from  $102^{\circ}$  to  $104^{\circ}$  F., and associated with vomiting and anorexia. In rare cases delirium and convulsions mark the onset. The fever lasts at the most a few days and may be so slight as to be overlooked. Upon examination of the affected part the paralysis is found to be flaccid, with a complete absence of reflexes in the distribution of the palsied muscles. This rule has no exception. The commonest location of the paralysis is in the peronei group. In the focal zone of the inflammatory process the loss of function will remain complete and permanent. In the perifocal zone where the nerve cells have been altered, but where restitution of function is possible, the mus-

cles will, after a few days, begin to regain function, until at last where a whole limb was at first affected the paralysis may persist in only a single muscle, a group of muscles, or the flexors alone. The paralysis is always of an atrophic, degenerating type (Fig. 177), and after a few days there will be present a slowing of contraction to the galvanic current, with the anodal-closing contraction greater than the cathodal; in unfavorable cases the failure to react to

Increased quantities of the galvanic current progresses, until after a few days there is absolutely no reaction. Where the involvement is extensive the limb fails to grow and remains much shorter than that of the opposite side (Fig. 178); the circulation is defective, the limb appearing cyanosed and feeling colder than the normal one. Where the paralyzing muscles to those paralyzed retain their function various deformities may result, due to the unopposed contraction of the normal muscles.

Sensation is not disturbed, as a rule, although in some cases the affected muscles may be tender to pressure. The sphincters remain intact. There is no disturbance of mentality.

FIG. 178



Acute anterior poliomyelitis.

**Diagnosis.**—In the acute stage this disease must be differentiated from simple congestive conditions of the cord, neuritis, rachitic pseudoparalysis, and cerebral palsies. There is a class of rare cases, two of which have come under my notice, where, after an apparently causeless attack, both lower limbs have suddenly become paralyzed with loss of reflexes, and with no disturbance of sensation. In both cases the prognosis of acute anterior poliomyelitis, with an unfavorable prognosis on account of the extent of the paralysis, was given. In both cases after

a week the paralysis entirely cleared up. I know of no way of making the differential diagnosis in these cases in the first few days, but if in paralysis of this extent the reactions of degeneration are not typically developed before the end of the week a favorable prognosis should be given.

The pseudoparalysis of rickets shows the following clinical picture: it affects both extremities and is at times associated with loss of reflexes, costal beading and enlarged epiphyses, soft muscles, sweating about the head, and a history of defective feeding. The tenderness of scorbutic infants with pseudoparalysis and the hemorrhagic gums will always disappear by dietetic treatment. From the cerebral palsies poliomyelitis can be differentiated by the flaccid type of the paralysis, with the loss of the reflexes and the absence of mental symptoms. Cerebral spastic paraplegia, which is most often mistaken for poliomyelitis, affects both lower extremities; the reflexes are exaggerated; the muscles are spastic, there are no reactions of degeneration; the condition is usually present from birth or very early life, and there is a history of difficult or prolonged labor or instrumental delivery.

The absence of pain and of tenderness along the nerve trunks, and the sudden onset will differentiate poliomyelitis from neuritis. I have found the greatest difficulty in diagnosing those cases in which the paralysis is localized to a single muscle or muscle group. It must be remembered that the only reflexes lost are those under the motor control of the paralyzed muscle. In a child of four years of age who began to walk on the heel a careful examination revealed a degenerating paralysis of the gastrocnemius and soleus on one side with an absence of the Achilles reflex on that side. All the other muscles of the extremity and all the other reflexes were perfectly normal.

**Prognosis.**—The prognosis of any individual case should not be made until after the case has been studied for a few weeks. The parents, however, may be assured that the resulting paralysis will not be so extensive as that at the onset. The more limited the paralysis the better the prognosis, but it is rare except in the most limited cases for a complete return of function. The prognosis is better when the paralysis affects the anterior distribution of the lower extremity than when it affects the posterior, and more return of function may be expected. The electric examination offers the best method of determining what muscles will regain function.

**Treatment.**—Absolute rest in bed for at least two or three weeks is necessary if we would limit the process to its primary destructive zone and give the cells in the congestive area a full chance to regain their function. If seen early in the febrile stage purgation should be established, and the fever reduced by a simple fever mixture and free sweating. While drugs have no curative agency the salicylates and belladonna have been much used. Dry or wet cups, blisters, and leeches have been applied over the lumbar area of the cord, with the idea of relieving the congestion, but I have never seen any benefit from their use. After the second or third week, during which time the nutrition of the child should



be carefully attended to, massage and passive movements should be instituted. While it is advisable to secure the services of a trained masseur, inasmuch as it will be often necessary and advisable to keep up this treatment at least a year, and often for two or three years, where the results justify continuance, some member of the family can usually be taught the underlying principles of massage so as to produce excellent results. Except in the very well-to-do it has been my rule to start the treatment with a trained masseur and have the most interested and suitable person in the family taught the necessary manipulation, and when a sufficient degree of proficiency is acquired to continue the home treatment. The idea in the treatment is to have a well-nourished muscle fibre ready to take up the function as soon as sufficient nerve power returns to the affected cells. Care must be used to prevent deformities, and is best obtained by passive movements given with massage. While electricity is a valuable adjunct in keeping the muscles in good condition, it is not necessary where proper massage can be secured. That current should be used which secures the best contraction with the least degree of pain and discomfort and with the least quantity of current. Gymnastic exercises if well regulated are valuable after the above treatment is well established. Where a muscle can be made to do its work without the assistance of apparatus the latter should not be employed. Where the paralysis, however, is such that it will be necessary to aid in securing rigidity of the limb to overcome a marked toe-drop or lateral deviation of the foot, a simple, light, mechanical appliance will be indicated. The more simple and lighter the apparatus the better, and it will not be necessary in all cases to secure either the services of an orthopedist or instrument maker to secure good results. An elastic band applied to the shoe and to a garter below the knee in such a way as to take up the function of the paralyzed muscles will be much more comfortable and serviceable than a complicated metal brace. A careful study of the muscles affected and of the mechanism of their action will decide in any individual case whether special apparatus will be necessary. In any event the greatest care in supervision on the part of both the attending physician and the orthopedist should be given in order to cultivate any returning power, to supply any increasing deficiency, or to correct any developing deformity. In cases coming under observation late it may be necessary to do a *tenotomy* in order to secure proper position before braces can be applied. In selected cases excellent results may be obtained by the transplantation of tendons. When a single muscle or allied group of muscles are paralyzed and the opposing muscles, or even muscles with a similar function, be intact, the tendon may be divided and either half of the tendon or the entire tendon implanted on that of the paralyzed muscle. Thus one of the tendons of the common extensors of the leg or even half of this may be attached to that of the anterior tibial when this is paralyzed. The tendon of the soleus or the split tendon of the tendo Achillis may be implanted upon the peroneus longus et brevis to restore function in this distribution. In the upper leg distribution the flexors of the leg may be transplanted to the tendon

of the quadriceps with good results. Similar results may be obtained in the upper extremity. The transference of function of flexor muscles to that of extensors or the reverse is established without difficulty and with little effort and training on the part of the patient. Recently Spiller has divided the nerves in a longitudinal direction and implanted half of the nerve going to a normal functioning muscle such as the common extensors of the leg to a paralyzed anterior tibial. It is, however, too soon to say how much value this method will have. Theoretically it should give better results than implantation.

### ACUTE MYELITIS.

Inflammatory conditions of the substance of the spinal cord, Myelitis, may be divided according to their course into acute, subacute, and chronic forms. The disease may be limited to one part of the cord or may be very extensive.

**Etiology.**—An infection of the spinal tissues during or following one of the acute infectious fevers is the most common cause of the disease. It has been known to follow smallpox, typhoid fever, dysentery, gonorrhea, syphilis, pneumonia, influenza, malaria, tonsillitis, and septic processes such as cystitis, pyelonephritis, felons, abscess of the antrum, and endocarditis. It results not infrequently by extension from purulent conditions of the meninges in epidemic cerebrospinal meningitis and tuberculous meningitis. Localized abscesses in the bones of the spine may rupture into the spinal canal, with the production of a transverse septic myelitis. Abscesses without the spine may extend along the nerve sheaths and produce a septic infection of the cord. Traumatism from stab wounds, bullet wounds, fracture of the spine, severe over-exertion, and septic infection following operations on the spine are recognized causes. Traumatism without rupture of the overlying tissues may lead to multiple punctate hemorrhages, which in turn act as irritants and lead to an overgrowth of the supporting tissue of the cord, presenting the manifestations of chronic myelitis. Extensive hemorrhage may occur, with destruction of cord tissue. I have reported a case of complete destruction of almost the entire dorsal cord from extensive hemorrhagic extravasation following severe traction on the lower extremities at birth. (See article on Spinal Hemorrhage.) Subacute myelitis is a common condition in Pott's disease, due to pressure on the cord from the thickened meninges. Alcohol, lead, mercury, and phosphorus have been blamed for disseminated areas of myelitis. Undoubted cases develop after exposure to cold, but whether the cold acts in lowering the resistance to other infections or acts upon some other underlying intoxication has not been determined. While the disease may occur at any time of life it is less frequent in childhood than in adult life.

**Pathology.**—The pathological process may be limited to one or two segments of the cord, may extend upward or downward to the rest of

the cord, or may be a diffused process with disseminated lesions about the vessels, affecting in an irregular way portions of or the entire cord. The membranous covering of the cord may be reddened and injected or may appear perfectly normal. On section of the affected areas the cord is of a soft, creamy consistence; the gray matter cannot be differentiated from the white and the cord substance is either of a red, injected color or, if the disease has existed for some time, irregular areas of yellow may be admixed with the red. If the process be localized, secondary degeneration occurs in the posterior column and the direct cerebellar tract from the point of the lesion to the medulla, and degeneration of the crossed pyramidal motor tracts and the direct pyramidal tracts below the point of lesion to the sacral cord. On microscopic examination the bloodvessels are congested throughout the entire cross-section of the cord, capillary hemorrhages are present in the gray matter and, at times, in the white substance, and a very marked and extensive accumulation of small round cells takes place about the vessels or infiltrates the entire area of section. The nerve cells of the anterior horn are found in all stages of degeneration. The evidence of intense destruction of the cord tissue is shown by the extensive change into fat when the sections are stained by osmic acid. The cells of the neuroglia supporting tissue undergo active proliferation in intense cases, and supply the area of the destroyed nervous elements when healing takes place. These cells may act with the leukocytes as scavengers for the removal of the destroyed nervous tissue. In the disseminated form small focal areas of perivascular round-cell accumulation will be found scattered here and there throughout the cord. In such cases the process is more likely to run a subacute or chronic course, and is more apt to be followed by sclerosis of the cord tissue with less active destruction of the cord elements than in the acute localized form. The intoxications, as a rule, lead to a slow overgrowth of the neuroglial tissue, without acute manifestations.

**Symptomatology.**—In the acute form, and especially that form due to septic infections, the disease develops rather suddenly, with fever varying from 100° to 104° F. Pain in the back may be slight or very intense, and referred to those areas supplied by the portions of the cord affected. There is marked tenderness to pressure on the back. Hot applications are poorly borne over the affected area. Evidence of disturbance of function of the cord are present very early, and depend upon the portion of the cord affected. If the process be localized, as it most frequently is, to the dorsal part of the cord, all motor impulses coming from the brain are interrupted at this point, and a paralysis of the legs, bladder, and rectum, and, if the lesion be sufficiently high, of the abdominal and spinal muscles results. All the sensory impulses (pain, touch, temperature, and muscular sense) are interrupted at this point in their course from the periphery to the brain and are not received. In other words, there is a complete loss of sensation in the lower extremities and of the trunk up to the upper level of the lesion. At this level the skin is hyperesthetic, due to the irritation of the nerve



fibres in the peripheral zone of the inflammatory process. If the process is limited to the dorsal cord, and the lumbar enlargement supplying nutrition and reflex function to the lower extremity remains intact, the reflexes of the legs will be increased, there will be ankle clonus, and upon irritation of the sole of the foot the toes will be extended instead of becoming flexed. (See p. 861.) The disturbance of the bladder from a lesion in this area will be a retention of urine followed by overflow—the incontinence of retention. This is due to the contraction of the sphincter, which retains its normal motor power, increased by the increase of reflex excitability, the retention of the reflexes, and the loss of volitional control from the interruption in the dorsal region. In lower lesions affecting the lumbar and sacral cord the sphincter becomes paralyzed, the reflexes are lost, and there is incontinence of urine without retention. The same is true for the rectum. In low lesions affecting the lumbar cord the paralysis is confined to the legs, and is of a flaccid type, with loss of reflexes, atrophic degenerations of the muscles, and loss of sensation to the upper limit of the lesion, which in some cases may be at the hip or in others as high as the upper limit of the inflammatory processes in the dorsal cord. In low lesions the loss of trophic influence due to the destruction of the anterior-horn cells commonly results in extensive bed-sores, which add a new source of infection and become a most serious complication.

If the process be localized to the cervical lesion of the cord, as is not infrequently the case in Pott's disease, there is a paralysis of the arms, flaccid in type, with loss of arm reflexes, due to a disturbance of the reflex mechanism localized to the cervical area. The biceps jerk and triceps jerk are absent. The paralysis in the arms is a degenerative atrophic type. The paralysis of the lower extremity, the involvement of the bladder and rectum, and the retention of the reflexes are the same as that of the dorsal lesions first described. In cervical lesions the entire body is anesthetic up to the neck.

In the severe cases with bed-sores and cystitis the temperature range is irregular, the tongue is dry and coated, the patient becomes delirious, and death ensues either from uremic or septic intoxication. In the less severe cases the inflammatory symptoms subside after a few weeks, and the patient either may remain completely paralyzed, with the development of contractures in parts below the lesion, or there may be partial or complete recovery of function, depending upon the extent to which the spinal tissues were destroyed. Recovery of function may take place in some of the muscles of an affected extremity, while others remain partially or completely paralyzed and atrophic. In lesions above the lumbar enlargement it is the exception for recovery to take place without some stiffness of the gait and loss of power.

In the subacute and mild acute cases sensation and motion may be only partially interfered with. Occasionally I have seen in cases associated with complete or only partial loss of power a hyperesthesia of the skin, followed only late or not at all by anesthesia. In the diffuse disseminated form all four extremities may be mildly affected with partial



loss of power and sensation, or the clinical picture may be the same as that above described.

**Diagnosis.** In the case above referred to in the etiology of extensive hemorrhage due to traction on the extremities an interesting case for diagnosis was presented. The attending obstetrician was accused of causing the paralysis and suit was threatened for malpractice. Immediately after birth it was noticed that there was complete paralysis of the lower extremities. When the child was stripped a flaccid, protuberant condition of the abdomen was presented. The distended bladder could

FIG. 179



Transverse myelitis. Paralysis of thorax, abdomen, and legs.

be seen as a spherical tumor rising almost as high as the umbilicus. Sensation was lost in the lower extremities and the trunk as high as the fourth dorsal vertebra. The reflexes of the lower extremity were present and prompt. There was no wasting of the lower extremities, nor were there reactions of regeneration to the electric current. This fact together with the incontinence or retention of urine led us to assume that the lumbar enlargement of the cord was normal, and that there existed a complete destructive lesion in the dorsal cord. The body above the waist was perfectly normal. A diagnosis of hemorrhage

into the cord at birth or shortly before birth after the spinal cord had been fully developed was made. This diagnosis was fully confirmed by autopsy, which showed an extensive hemorrhagic extravasation destroying a large portion of the dorsal cord. (See Fig. 179.)

**Prognosis.**—In severe cases the necessity of evacuating the bladder by means of a catheter exposes the patient to such risks from external infection, due to the lowered resistance, that it is always an important factor in leading us to make a guarded prognosis. The same may be said of bed-sores; and when with extensive bed-sores the temperature becomes high and irregular, in spite of local treatment, a fatal outcome may be expected. In early infancy the prognosis is more unfavorable than in later life. The prognosis as far as recovery of function is concerned depends on the extent of the primary loss of function and the course of the disease. If the inflammatory lesion persists for several weeks with some fever, and the restitution of function during this time does not become evident, the resulting paralysis will in all probability be persistent. If the inflammatory symptoms rapidly subside, even though the paralysis at first may be complete, fair restitution of function may take place. The prognosis must in all cases be a matter of study of the individual case.

**Treatment.**—The sooner the patient is placed at absolute rest in bed the better. A careful search should then be made for any underlying sepsis or intoxication, and this as far as possible removed. If there be evidence of syphilis a course of mercurials or of the mixed treatment should be immediately begun if this is considered to be the cause of the disease. It is better in all severe cases to employ from the beginning a water or air mattress. The air mattress should be perfectly smooth, and the air free in the mattress so that the surface of the body will rest in uniform pressure. The air mattress used in camping, divided into compartments and with an irregular flat surface, should not be employed. A proper air mattress, with scrupulous cleanliness and oversight that the bed-clothing should be kept dry and evenly spread without wrinkles, is the best method of preventing bed-sores. In retention of urine where catheterization is necessary, the greatest care and cleanliness should be observed; in incontinence of urine a bed urinal may be employed, or wads of antiseptic cotton frequently changed may answer the same purpose. Careful attention to the above details by a conscientious trained nurse is of the utmost importance in the treatment. Frequent washing of the back with an astringent solution, such as alum and alcohol, will assist in keeping the skin in good condition. Reddened areas of the skin, the forerunner of bed-sores, should be carefully removed from pressure by the air ring or rings of cotton carefully applied. The skin should be kept clean and the red areas painted with nitrate of silver 0.65 gm. (10 gr.) to 1.3 gm. (20 gr.) to 30 c.c. (one ounce) to harden the skin. If bed-sores have formed, pressure should be likewise relieved, the ulcerated surface frequently cleansed with hydrogen peroxide, followed by a weak carbolic or other antiseptic solution, and a constant wet dressing applied. Where any tendency to healing is

shown, the edges may be touched up with strong solutions of nitrate of silver and ointments applied. I have found the use of a prescription of

R—Cocaine . . . . .	0.06 gm.	(gr. j).
Menthol . . . . .	0.30 gra.	(gr. v).
Petrolatum . . . . .	30.00 gm.	(℥j).
M. et ft. unguentum.		

useful. The general nutrition of the patient should be carefully attended to and a good nutritious diet employed.

Local applications to the spine do little good. Long ice-bags or in other cases hot applications relieve the pain. The spasmodic contraction of the legs in dorsal lesions may be relieved by hot applications and the use of the bromides internally. Internal medication during the acute process, except in the cases due to syphilis, gives little result. The salicylates are frequently used in the infectious cases and may do some good.

During convalescence from the acute process the patient should be carefully guarded from attempts at walking or other use of the muscles. I have known cases otherwise doing well to develop serious symptoms from the jar of falling or in riding, as in one case where it was necessary to remove the patient in a carriage over city streets several weeks after convalescence had begun. The primary return of power may be expected to be more or less interfered with by the contraction of the inflammatory tissue and the secondary degeneration above described. To lessen this secondary rigidity and to prevent contractures are the main objects of treatment during convalescence. No active motion should be permitted for at least a month. Passive movements should then be begun and gentle massage employed. Ordinary massage, as a rule, unless carefully performed by a skilled operator, leads to spasmodic contractions of the affected muscles. Passive movements carried out with the patient in a hot bath and gentle massage under hot water give the best results. In using the hot bath for this purpose care should be taken to protect the patient from cold. When the inflammatory process affects the lumbar or cervical enlargement and there is wasting of the muscles, massage, electricity, and the use of mechanical appliances as directed for the paralytic conditions of poliomyelitis should be employed.

It may be necessary to overcome the contractures by tenotomy of the hamstring or Achilles tendons. When the angle of contracture is marked, gradual straightening of the extremity should be employed in preference to the rapid method in vogue among surgeons. In a case of mine, where the latter method was employed, a degenerative paralysis of the legs below the knee resulted from a too forcible stretching of the perineal nerves. Section of the Achilles tendon relieves the spasm of the calf muscles and is beneficial in controlling a persistent ankle clonus—a very troublesome condition which seriously interferes with the gait of the patient. Transplantation of tendons for the paralyzed and atrophic muscles may be employed as in poliomyelitis.



## POTT'S DISEASE.

Potts Disease is of importance in connection with diseases of the spinal cord, because of the various mechanical, inflammatory, and degenerative changes it produces.

While Pott's disease may occur at any time of life it is much more common in childhood. The disease is a tuberculous process affecting the bodies of the vertebrae and occurs in children of a tuberculous or scrofulous diathesis. Traumatism is an important determining factor, and need only be slight. This disease is here considered with reference to the changes it produces in the spinal cord.

**Pathology.**—The disease of the bodies of the vertebrae (one or more vertebrae may be involved) results in a displacement of the bodies of the vertebrae one upon the other, with a resulting deformity of the spine. The extent of the deformity depends to a great extent upon the part of the spine affected and the age of the patient. In growing children, and before the bones of the spine have set for their adult function, the deformity is much more common and much more extensive than it is in adult life. When the process is localized to one or two vertebrae, a slight angular kyphosis is presented. In lesions of the cervical region it may be necessary to carefully search for any deformity. From a slight angular kyphosis all grades are seen—to extensive arching and irregular deformity. The result of the deformity is to produce a narrowing of the spinal canal. If the narrowing be marked it may lead to pressure on the spinal cord, which becomes flattened out and diseased at this point. This narrowing of the spinal canal is in some cases so marked as to leave only a small passage for the cord. The cord may be only one-fourth of its normal diameter. The compression of the cord and the resulting symptoms are accentuated by the development of a pathological exudate on the outer surface of the dura mater (external pachymeningitis). The thickening of the meninges is due to the irritative bone process. In cases with little deformity this may be so marked and extensive as to cause marked compression of the spinal cord with little narrowing of the spinal canal. (See Figs. 181, 182.)

In rare cases a localized abscess may form in the bone and suddenly rupture into the canal, with complete local destruction of the spinal cord. In a case with no deformity, in which the bone lesion was not suspected, the symptoms developed suddenly, with evidence of complete destruction of the cord in the upper dorsal area. A diagnosis of hemorrhage into the cord was made. At the autopsy an abscess cavity in the body of one of the vertebrae was found to have ruptured into the spinal cord.

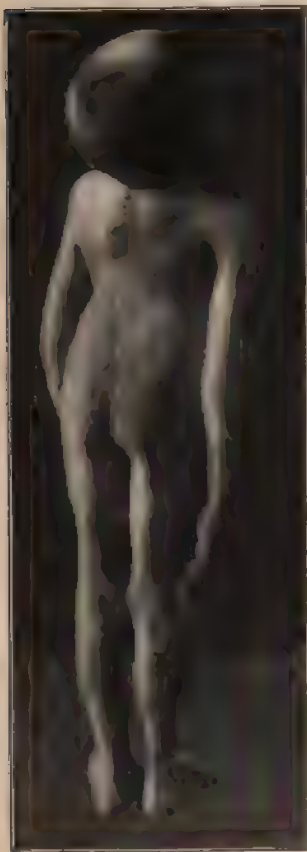
While the bone process is usually localized to a single area of the spine, double lesions are not infrequently met with.

**Lesions of the Spinal Cord.**—The extensive inflammatory exudate on the dura is shown at autopsy by strong adhesions at the point of disease. The cord at this point may be softened, edematous, or, in slowly progressive



ssive cases of long duration, may be of normal consistence but flattened. Above and below the point of pressure secondary degeneration the cord is marked. On microscopic examination the changes vary in a slight edema with congestion in the mild cases to extensive destruction of cord tissue, capillary hemorrhage, and round-cell infiltration about the vessels. An acute, terminal tuberculous inflammation

FIG. 180



Cervical Pott's disease.

FIG. 181



Dorsolumbar Pott's disease.

n of the membranes of the cord (the pia mater and arachnoid) or a localized chronic plastic exudate on the inner surface of the dura may be present.

**Symptomatology.**—In this disease the symptoms referable to the spine develop insidiously. They may occur immediately after a traumatism, or a period of weeks or even months elapse. The spine becomes more rigid, there is a certain stiffness to the body movements, and

localized pain and tenderness are presented. Subjective symptoms may be, however, entirely absent, and the very gradual development of the spastic paralysis of the lower extremities may be the first evidence of the disease process. Inasmuch as the caries affects the dorsal and cervical regions in most cases, the paralysis is spastic in type, with an increase of the reflexes, ankle clonus, and the Babinski reflex.

In the early stages one leg may be more paralyzed than the other. There may be no disturbance of sensation in the mild cases, or there may be complete anesthesia in the severe cases up to the level of the lesion. In such cases the bladder and rectal functions will be disturbed as in cases of myelitis. The inflammatory process in the dura mater may produce irritation of the nerve roots and cause intense pain in their distribution. Slight irritation of the roots gives rise to increased sensitiveness to touch and pain impressions. The distribution of the pressure on the spinal cord may be such as to cause a loss of some forms of sensation with the retention of others. Thus sensation for pain and temperature may be lost, and sensation for touch retained and normal. If the cervical part of the spinal cord is affected all four extremities are involved; a flaccid paralysis with wasting and degeneration of the muscles in the arms, and a spastic paralysis in the legs. In cervical lesions above the cervical enlargement the paralysis of all four extremities may be spastic in type, and the sensory disturbance affect the whole body with the exception of the head. Bed-sores are sometimes present. In pressure lesions affecting the lumbar enlargement of the cord, the paralysis of the lower extremities is flaccid in type, with loss of the reflexes, wasting of the muscles, bed-sores, and incontinence of urine.

**Diagnosis.**—The diagnosis of Pott's disease in children is not a difficult matter if the rules for diagnosis of nervous conditions of childhood, laid down at the beginning of the chapter, are observed. Even in the very early cases, where the disturbance of the motor function is very slight, an examination of the naked child will show the limitation of the movement of the spine and any slight deformity when the child bends the body. Percussion over the spine with the index finger placed over each successive vertebra and a fairly strong percussion tap made on this finger will reveal a sensitive area at the point of disease. In advanced cases there is usually no difficulty on account of the very evident deformity. I have seen, however, in an adult an aneurysm produce erosion of the vertebra and deformity of the spine sufficiently marked to be mistaken for Pott's disease.

**Prognosis.**—Prognosis in any individual case will depend upon how far the disease of the bone can be controlled. In mild cases if the proper treatment can be carried out the prognosis for return of function in the course of a few months is good. Prognosis for the return of function of the cord, even in those cases where there is marked pressure and flattening out of the cord, is not altogether unfavorable. Even a very thin, narrow band of spinal cord has been found to transmit impulses which in cases due to other lesions would not be expected. This is

due probably to the gradual development of the pressure and the accommodation of the nervous tissues to the new conditions.

**Treatment.**—The treatment of the spinal lesions is the removal of the bone disease. Whether this be obtained by rest in bed, with extension and nutritive measures, or whether surgical means are employed, the result should be obtained as soon as possible in order to relieve the pressure on the cord. The spine should be placed absolutely at rest either by the head extension above referred to or a plaster-of-Paris jacket. Fresh-air treatment with overfeeding by milk and eggs—preferably carried out, especially during the summer months, at some seaside resort, where the bed of the patient with its extension apparatus intact can be wheeled to a porch in the fresh air and sunshine secured most of the day—will give the best results. Direct exposure of the back and the trunk to the sun has given valuable results. This may be also carried out with a little extra trouble at home. It should be remembered, as far as active surgical procedures for the relief of the bone condition or the removal of the exudate about the spinal cord is concerned, that children react poorly to operative insults to the nervous system, and there is always the possibility of producing a blood infection with miliary tuberculosis by even slight operative procedures. The same is true of forcible extension of the spine, with the idea of forcing the bones back to a straight position at a single sitting. Much better and safer results are secured by more conservative measures.

The treatment of the paralysis, the wasting, contractures, and bed-sores is the same as that suggested for Myelitis and Poliomyelitis.

Medicinal treatment apart from the alterative tonics is of little value. When there is no lung involvement the iodides and mercurials have been given with reputed benefit.

#### TUMORS WITHIN THE SPINAL CANAL.

Tumors of the spinal cord or its meninges, while rare at any time of life, are especially rare in childhood. Of the fifty cases of tumor of the spine collected by Mills and Lloyd, 14 per cent. were under twenty years of age, four before the age of ten, and three between ten and twenty. The most common forms of cord tumors in childhood are syphilitic and tuberculous. Gliomata and cystic tumors sometimes occur. The tuberculous tumors may be multiple and can occur in the same case with a Pott's disease. In a boy of eight who died from a miliary tuberculosis following an operation for caries of the foot, two tumors tuberculous in nature in the sacral portion of the spinal cord and cauda equina were found. In another case a tuberculous tumor of the meninges, which completely infiltrated and destroyed the spinal cord in the dorsal urea, was associated with a tumor the size of an olive in the cauda equina. Tumors either of the cord or of the meninges cause destruction of the cord by slow invasion or by pressure. Above and below this point secondary degeneration occurs.

**Symptomatology.** The symptoms of tumors within the spine develop gradually and are progressive. Tumors beginning in the meninges produce intense pain by involvement of the posterior roots, and the pain is referred to the distribution of the roots involved. The pain in tumors of the lumbar enlargement is referred to the legs, in the dorsal enlargement to the chest, and in the cervical enlargement to the arms. There may be some tenderness on percussion at the seat of the tumor. Involvement of the anterior roots by the tumor process produces muscular jerks very early, followed by paralysis and muscular atrophy. When the tumor begins within the spinal cord the symptoms of root irritation, lancinating pains, atrophy, etc., develop late. The other symptoms of tumors of the spinal cord are due either to compression or to destruction of the cord tissue, and in this respect will not differ in results from those of a local myelitis at this same area. These symptoms will, however, develop very gradually and will not be associated with the symptoms of inflammation of that process. When the tumor begins on one side of the cord the symptoms at first are referred only to this distribution. Thus, given a tumor above the cervical enlargement, there will at first be a constricting pain about the neck, a dull pain over the cervical processes, and a progressive loss of power of the arm and leg of the same side as the tumor. The paralysis of the arm and leg will be spastic in type or at least associated with increase of the reflexes, with ankle clonus and the Babinski reflex. When only one-half of the cord is involved the sensory fibres of touch, pain, and temperature from the opposite side of the body, which have crossed over as soon as they have entered the cord, will be obstructed at this point, and there will be an anesthesia to all these forms of sensation on the side opposite to that paralyzed up to the point of lesion. The sensory fibres on the same side as the tumor, having crossed over upon entering the cord, find an unobstructed path through the unaffected half of the cord to the brain, and sensation on this side will be normal. As the disease progresses and more than half of the cord is involved the loss of power gradually affects the arm and leg of the opposite side, and sensation over the entire body is affected to complete loss. When this stage is reached there is incontinence of urine and feces and a tendency to contractures in the paralyzed muscles. When the tumor affects the cervical enlargement flaccid paralysis of the arm or arms will be found, associated with wasting as soon as the destruction of tissue in this area is complete. The paralysis below this point will remain spastic as before. When the lumbar enlargement is involved the paralysis is confined to the lower extremity, and becomes flaccid and wasting in type when the destruction of the cord tissues is completed. There will be incontinence of urine and feces. Sensation may at first be lost here only in the opposite leg, but later may affect both legs.

**Diagnosis.**—From Pott's disease tumors can usually be differentiated by the deformity, the predominance of bone pain, and the evidence of bone disease, and in cases without deformity by an x-ray examination. The loss of power in both conditions comes on slowly, but there is not



which evidence of complete loss of power in caries as in tumor. Tumors, as a rule, run a more rapid course.

From myelitis a tumor can usually be differentiated by the slowness of onset of the latter, with absence of inflammatory symptoms and the predominance of pain; whereas, in myelitis the onset is rapid, there is less pain, and rapid destruction of function. Restitution of function after myelitis after the acute symptoms have subsided is an important factor in diagnosis. Tumor can be differentiated from neuritis or multiple neuritis by the more rapid onset of the latter, the tenderness of the nerves and muscles, and the loss of reflexes. From cerebral lesions tumors of the cord can be recognized by the localization of the symptoms below a certain area of the cord, the absence of involvement of the face, or mental functions, and the involvement of the bladder and rectum.

The diagnosis of the character of the tumor can only be made in a presumptive way from the associated symptoms. Thus in a case where there is tuberculosis elsewhere in the body, a family history of tuberculosis, and no evidence of bone disease, a presumptive diagnosis of a tuberculous tumor can be made. If there is a history of inherited acquired syphilis, or if there is evidence of active syphilis elsewhere in the body, or a previous history of such, a gumma is diagnosed. If there is a history of echinococcus infection elsewhere in the body, and there is a variation in the intensity of the pressure symptoms with more destructive symptoms, a cyst may be diagnosed. If these forms of tumors be excluded a glioma or sarcoma may be present. The determination of the character of tumor is always more or less guesswork and unsatisfactory.

**Prognosis.**—Prognosis in all forms of tumor, with the exception of meningeal and of simple cysts, is unfavorable. In gumma and in other forms of syphilis of the cord the prognosis will depend entirely upon how early the treatment is begun and how vigorously it is carried out. When destruction of tissue has already taken place little result may be expected from treatment. In all other forms of tumors, while they occasionally yield to medical treatment, the only hope for the patient is an operation. When it is remembered that tumors of the spinal cord in children are very rare, and that early life is a deterrent to most operations for a serious operation on the nervous system, little can be gained from statistics. Statistics upon operations on the nervous system, whether considered here or elsewhere in this chapter, should not be given too much weight, because in rare operations we are much more likely to find a successfully treated case placed on record than one in which the results are bad or where there is a fatal outcome.

**Treatment.**—When a tumor is diagnosed the question of surgical procedure should be immediately considered. Two or three weeks may be devoted to the administration of mercury and iodide, and if no positive results are secured and the case is otherwise favorable, an operation should be done. While the results of operation are too often unsatisfactory it is the only hope, after medical treatment has been tried, in

keeping the patient from a fatal termination or, at the best, a life of chronic invalidism. Operations for simple cystic formations of the meninges pressing on the spinal cord may be completely successful if the patient withstands the shock of the operation. A case of this kind was recently reported by Spiller.

#### TRAUMATIC INJURIES OF THE CORD.

**A. Concussion of the Spinal Cord.**—This subject has given rise to so much discussion in connection with the subject of railway injuries, in a medico-legal relation, that a definition of exactly what is meant is quite necessary. The molecular changes of the older writers are too hypothetical for consideration. A sudden traumatism without other injury of the spine or cord may give rise to capillary extravasations of blood, degeneration of the sheaths of the nerves, and a secondary overgrowth of neuroglial tissue, either localized to the cervical enlargement of the cord or diffusely distributed throughout the entire cord. Such results may be seen in the spinal cords of patients dying from intercurrent diseases after falls from a height or after railway accidents. Identical lesions have been experimentally produced in the lower animals. The results of this condition apart from the shock produced are as follows:

There may be at first a paralysis or marked weakness of all four extremities, which is recovered from in the course of a few days or weeks, and is followed by a semispastic condition of the muscles with awkwardness and stiffness of movement and a marked excitation of all the reflexes. In severe cases in the early stages all forms of sensation may be lost, or only sensation for pain and temperature. In other cases there is no disturbance of sensation; in still others there is anesthesia limited to one side of the body, due to hysteria. From the minor ultra-microscopic changes of the nerve fibres and cells and slight capillary hemorrhage to extensive destructive hemorrhages many gradations may be observed.

**Prognosis.**—This will depend on the extent of the damage to the cord. In mild cases complete recovery takes place in a few months. In other cases permanent loss of power and wasting in the arms occurs.

**Treatment.**—Rest in bed, with massage, galvanism and graduated exercises are indicated.

**B. Hemorrhage of the Cord.**—There may be a hemorrhage, local or extensive in character, into the cord tissues, with partial or complete destruction, or the hemorrhage may surround the cord. In children this condition is practically always due to traumatism. The hemorrhage is usually the result of a fall of a considerable distance, where the child lands in such a way, either on the feet, the shoulders, or all four extremities, as to dissipate the force without fracture or dislocation of the spine. The vessels in the gray matter of the spinal cord are so poorly supported by surrounding tissue that a rupture occurs, and a

cal hemorrhage confined to the gray matter sufficiently extensive to involve the white matter or to destroy the entire cross-section of the cord at this point, or in rare cases several segments of the cord, is presented. In the case above referred to in myelitis an extensive area of the dorsal cord was destroyed by hemorrhage into the cord due to action on the feet at birth. Practically the same causes operate in the production of meningeal hemorrhage.

**Symptomatology.**—The onset is sudden at the time of the accident. If the hemorrhage be confined to the gray matter of the cervical enlargement there is at first complete paralysis of all four extremities, with loss of sensation up to the upper border of the lesion. After a few days the edema of the cord tissue surrounding the hemorrhage, which has led to the pressure causing the symptoms of the transverse lesion at this point, subsides and function rapidly returns in the lower extremities. One or both arms remain partially or completely paralyzed, with loss of reflexes and wasting in the paralyzed parts. This is due to the destruction of the anterior horn cells by the hemorrhage. While in some cases sensation completely returns, in other cases the pain and temperature fibres are interfered with either in their course through or in the neighborhood of the gray matter, and a loss of sensation to pain and temperature impressions, persists for some time, while sensation for touch remains perfectly normal.

If the entire segment of the cord in its cross-section be destroyed there is a complete loss of all function below the point of lesion. Paralysis of the arms will be flaccid and wasting; paralysis of the legs spastic with increased reflexes, the Babinski reflex, ankle clonus, and incontinence of retention of urine will be present.

In hemorrhage into the lumbar enlargement the paralysis is confined to the legs, is of the flaccid, degenerating type, with loss of reflexes and incontinence of urine. In a case with a small hemorrhage into the thoracic cord there was paralysis of the calf muscles, atrophic ulcer of the sole of the foot, incontinence of urine, and a saddle-shaped area of anesthesia of the posterior surface of the thighs, all of which developed suddenly after great overexertion.

**Hemorrhages into the meninges** of the cord are due to the same causes as those of hemorrhages into the cord, and produce symptoms of pressure on the cord which rapidly subside. A sudden paralysis of motion and sensation below the area of hemorrhage, with some lancinating pain due to the irritation of the roots, develop at the time of the hemorrhage. These symptoms rapidly disappear and may leave no sequelae, or there may be evidence of chronic irritation at the point of lesion due to organization of the clot.

**Treatment.**—The treatment follows the same lines as that described under Myelitis.

**Prognosis.**—This depends in a study of each case. When there is extensive loss of nerve tissue, very little return of power is to be expected. In meningeal hemorrhage rapid and complete return of function is the rule. Irritative symptoms sometimes persist. Traumatic

hysteria or neurasthenia may complicate the clinical picture and persist for a long time after the other symptoms have disappeared.

**C. Fracture and Dislocation of the Spine.**—The results due to both of these conditions are practically the same. In either case the cord is pressed upon and is crushed or completely destroyed. Any part of the spinal cord may be injured, the dorsal cord being most frequently affected. There is usually complete loss of function below this area. Paralysis is spastic in type, with increase of the reflexes and incontinence of retention of urine. The upper border of the lesion may be diagnosed by the determination of the upper limit of loss of sensation. The lower limit may be determined by the area of preservation of reflexes. Thus in a case of crush of the cord due to fracture, with extensive bone tenderness and crepitus in the lower dorsal area, the upper limit of anesthesia corresponded to the first lumbar segment, the preservation of the knee-jerk indicating that the third lumbar segment at least was functionally intact.

**Treatment.**—The only treatment is operation. The results of operative treatment in the large majority of cases of injury to the spine has given very poor results for the return of function after the relief of pressure. The spinal cord is usually so crushed that little result can be expected. Even where the restitution of nutrition of the intraspinal tissue is secured the restitution of function is usually a very slow process, and is a matter of months or even years until the maximum results are obtained. The treatment of the patient whether operation be attempted or not does not differ essentially or even in detail from that given above for myelitis. Suture of the spinal cord has been attempted in one case, but the return of power has been unsatisfactory.

#### SYPHILITIC DISEASE OF THE SPINAL CORD.

Syphilis of the Spinal Cord in children is usually the result of hereditary syphilis, but not infrequently cases occur of infection of children by the parents or accidentally from others. I have in mind a family in which four children and their mother presented evidence of active acquired syphilis from a drunken and dissolute father. In the acquired form of syphilis the disease may follow the type of syphilis of the nervous system in the adult. In the hereditary form there may be active syphilitic manifestations, or the resistance of the nervous system to external infections may be lowered. The gumma as a symptom of tertiary syphilis acquired or inherited has already been considered under the subject of Tumors of the Spinal Cord. The other conditions met with in the spinal cord are myelitis and meningomyelitis. The myelitis does not differ from that due to other causes, and may be acute, subacute, or chronic. There is, however, in these cases a more marked involvement of the bloodvessels and resulting endarteritis. It is usually a manifestation of secondary syphilis, although the chronic forms may occur in the tertiary stage. While the myelitis may occur



alone, it is usually associated with inflammation of the surrounding membranes of the cord. It is rare to have an inflammation of the meninges of the spinal cord without involvement of the cord tissue. To this combination the term meningomyelitis has been given.

**Symptomatology.**—The symptoms of an acute syphilitic myelitis localized to one part of the spinal cord do not differ from those due to other causes and described above. In the subacute and chronic forms, where there is an associated involvement of the membranes and the bloodvessels in an irregular way over extensive areas of the cord, the symptoms follow a rather irregular distribution, depending entirely upon the cord tissues involved. Inasmuch as the dorsal cord bears the brunt of the attack in the majority of cases, the most common manifestation of subacute and chronic syphilis is a paraplegia, spastic in type, developing rather slowly, but at times following evidences of a rather acute inflammatory process, and associated with increase of the reflexes and involvement of the bladder function. As the disease progresses the other tracts of the spinal cord are involved and irregular areas of loss of sensation on the trunk and the extremities are present. When the meningeal process extends to the lumbar enlargement some of the anterior roots are involved, and irregular atrophy of one extremity or some groups of muscles results. The reflexes in the atrophic distribution become diminished and are finally lost, while in the muscles which remain intact and spastic they are persistent and increased. When the process extends to the cervical enlargement there may be an atrophy of a single group of muscles or of one arm, and perhaps associated with a spastic condition of the opposite arm, or this arm may remain perfectly free, or there may be simply loss of power with atrophy affecting the muscles of the hand. Sensory disturbances may be present in the arms when the posterior roots become involved. Irritation of the posterior roots, transmitting the pain impressions from the periphery to the spinal cord, may result in constant or intermittent lightning-like pains referred to any portion of the body, depending entirely upon the roots affected. A careful history of the disease will reveal a continuity or regular sequence of the symptoms depending upon the pathological process, beginning in one area of the cord and extending in its irregular way along the meninges with the involvement of the underlying cord tissue. There is in these cases only an apparent irregularity and atypical arrangement of the symptoms, and if the case be studied with the extension of the pathological process in mind, and the anatomy and physiology of the cord involved be taken into consideration, the clinical picture can easily be understood and interpreted. It is neither right nor scientific to make a diagnosis of syphilis of the cord simply because the symptoms presented do not fit into the picture described for other diseased types.

**Diagnosis.**—The diagnosis must depend to a great extent on the rule of the extension of the process laid down above, and especially upon the history of the case and other evidences of somatic syphilis. The spastic form may be mistaken for the cerebral palsies of childhood, and specially for that form in which both legs are affected. The absence

of local atrophy, the increase of all the reflexes of the lower extremity, the absence of involvement of sensation of a spinal type, the history of difficult or prolonged labor, and the lack of other evidence of syphilis in cerebral palsies will make the diagnosis. Acute anterior poliomyelitis can easily be differentiated from syphilis by the absence of disturbance of sensation, of pain, and a normal condition of the reflexes of the rest of the body, with loss of reflexes in the area of paralysis. Even in those cases of poliomyelitis where there is more than one focus of inflammation and where an arm on one side and a leg on the opposite side may be paralyzed, or where an arm or leg on the same side may be affected, the acute onset of the disease and the absence of sensory or pain symptoms will usually be sufficient to make a diagnosis. In those cases where a gumma, a myelitis, and extensive involvement of the meninges are associated in a single case the mere combination of the three separate groups of symptoms will point to syphilis as the causative factor.

**Treatment.**—Mercury and the iodides give good results, when administered before destructive connective-tissue changes take place; when given late they are of little benefit.

#### DISSEMINATED SCLEROSIS. MULTIPLE SCLEROSIS.

While cases of Disseminated Sclerosis first come under observation during the second decade of life, there is little doubt that in a large number of cases not only are the symptoms present during the first decade of life, but may be present even at birth (Totzke).

**Etiology.**—The disease is commonly regarded as associated with the infectious diseases of childhood, but it may not follow until some years after an infectious fever. It may occasionally develop immediately after an injury. Oppenheim has insisted on the toxic nature of this affection and has directed attention to the influence of metallic poisoning as a factor in its production. The occurrence of the disease at birth and of cases found in succeeding generations has led some to consider it an hereditary type of disease. Direct heredity, however, is as rare in this as in other forms of organic nervous disease.

**Pathology.**—Irregular patches of sclerosis are found in almost every portion of the central nervous system. They are more frequent in the white matter of the brain, in the pons and medulla, and usually in a symmetrical way in the posterior half of the spinal cord. The areas of sclerosis, however, follow no definite rule of location and may occur anywhere. There is a certain vague relation in their early formation to the distribution of the bloodvessels. Microscopic examination shows in the early stages patches of sclerosis surrounding the bloodvessels with proliferation of the neuroglial cells, destruction of the myelin sheaths of the nerve fibres, but with a preservation of the axis cylinders in the sclerotic areas.

**Symptomatology.**—The symptoms presented are at first weakness of the lower extremities followed by a similar condition of the upper

extremities, with increase of the reflexes and a spastic gait. A characteristic intention tremor develops early. There may be no tremor of the hands at rest, but when some voluntary action is attempted a coarse tremor becomes so marked as to prevent the patient feeding or caring for himself. The speech about this time becomes affected and presents a slow, deliberate, tremulous, scanning quality. An examination of the eyes shows a marked oscillation of the eyeball from one side to the other on lateral movement. An examination of the eye fundus usually shows a marked pallor of the temporal side of the nerve head due to patches of sclerosis in the optic nerve or the optic commissure. The pupils are more or less contracted and react somewhat sluggishly to light and accommodation. In rare cases there may be no reaction to light, but reaction to accommodation is retained. The memory not infrequently becomes weakened and the other intellectual faculties may be likewise impaired. The combination of the intention tremor with nystagmus, scanning speech, and mental defect is characteristic of this affection. If to this be added irregular manifestations due to patches of sclerosis anywhere in the brain or cord a diagnosis can easily be made. When the lesions first develop in the cord the diagnostic symptoms may not develop for several years.

In one case a patch of dense sclerosis in the posterior columns led to a diagnosis of locomotor ataxia, which was changed after two years by another observer to ataxia paraplegia. This was due to an involvement of both lateral columns of the cord by irregular patches of sclerosis in the interval. A year later the loss of power in the lower extremities was so complete and the spasticity so marked, due to a lesion high up in the dorsal cord which involved the entire area of cross-section, that a diagnosis of spastic paraplegia was made by a third observer. The lesions in this case, which were so long confined to the spinal cord, had they been associated with the symptoms due to involvement of the cervical enlargement of the cord, the medulla, and the pons, would have led to an easy diagnosis. The examination of the eye-grounds in this case apart from the above symptoms would have shown the irregular areas of whitening of the disks due to patches in the optic nerves and commissure.

In some cases when the patches of sclerosis affect the anterior horns of the spinal cord atrophy of the muscles develops. The involvement of sensation is irregular and depends upon the patches of sclerosis in the spinal cord. Areas of anesthesia present at one time during the disease may disappear if the axis cylinders running through the sclerotic patches do not undergo complete degeneration. Paralysis of the cranial nerves also occurs, particularly of those nerves supplying the ocular muscles.

**Diagnosis.** Multiple sclerosis occurring in childhood and presenting the scanning speech, the nystagmus, the intention tremor, and mental defects is not likely to be mistaken for any other condition. There are cases, however, which have been diagnosed in the early stages as chorea or spastic spinal paraplegia. In such cases time aids in the diagnosis and the examination by the ophthalmoscope revealing changes in the optic disk as mentioned above will be of considerable value.



**Prognosis.**—Prognosis as far as life is concerned is good. Cases beginning in childhood rarely live beyond middle life. There is no hope for cure.

**Treatment.**—The treatment of a disease of a type recognized from the beginning to be incurable must be in the direction of making the patient comfortable. This is best secured by attention to the general hygiene, the avoidance of fatigue and prolonged hours of rest. The tremor is to a certain extent controlled by keeping the muscles in good condition, by massage, electricity (galvanism), and hydrotherapy. A tepid or warm bath associated with gentle massage gives the best effect. Many drugs have been used with negative results. The iodides, bromides, mercurials, and nitrate of silver are the favorites.

### ABIOTROPHIC DISEASES.

Gowers has recently used the term Abiotrophy to designate that condition of tissues in which there is an inherent defect or lack of vitality. This is manifested by an early degeneration or loss of function of the tissues affected. The class of diseases referable to the nervous system which we shall consider as belonging to this group are: *a.* Hereditary ataxia (Friedreich's ataxia). *b.* Hereditary spastic paralysis. *c.* Muscular dystrophies.

#### HEREDITARY ATAXIA.

Hereditary Ataxia, or Friedreich's Ataxia, is essentially a disease of childhood. Friedreich in describing this disease in 1863 considered it a juvenile form of locomotor ataxia, and called attention to its congenital origin and to the fact that it affected several members of the same family.

**Etiology.**—Friedreich's disease always occurs in early life (Fig. 182). It is usually fully developed before the second decade. Cases developing after this period are always open to the suspicion of belonging to some other disease group. Of the 143 cases collected by Griffith 15 occurred before the age of two years, 39 between the second and sixth year, 45 between the sixth and tenth year, 20 between the eleventh and fifteenth, 18 between the sixteenth and twentieth, and 5 between the twentieth and twenty-fifth year; 86 were males and 57 females. Some cases have followed the infectious fevers, but they are factors only in so far as they develop an inherent abiotrophy. Oppenheim has considered an inherited syphilis to be a factor; he has, however, found few to agree with him.

**Pathology.**—Grossly the spinal cord is smaller than normal. The microscopic examination shows an extensive degeneration in the posterior and lateral columns. The degeneration is associated with an extensive sclerosis in the columns of Goll and Burdach, more marked in the former, and a lesser grade of sclerosis in the crossed pyramidal motor tracts, and of Clarke's column of ganglion cells in the posterior gray



horns. The degeneration of the spinal cord extends as far as the medulla. Atrophy of the posterior roots and of the peripheral nerves has been described. Recent careful examinations of the rest of the spinal cord have shown a defective development (diminution of the number of fibres in areas not affected by the sclerosis). There are also fewer ganglion cells in the anterior and posterior horns than in normal cords. Marchi has stated that secondary changes are present in the cerebellum. There is therefore a marked degeneration with sclerosis affecting both motor and sensory paths and evidence of defective development of the other cord tissues.

**Symptomatology.**—The disease may be congenital and an absence of motor power be present from birth. These children never develop the power to stand or walk and eventually present the same clinical picture as found in those who acquire the disease later. As will be seen from the tables quoted most cases begin between the fifth and tenth years of life. A careful history will usually show some manifestations prior even to this time. When the child learns to walk it is often found that he is unsteady on his feet with an awkward staggering gait. If he has already learned to walk an ataxia of the lower limbs is first manifested; the gait is unsteady and swaying, the legs spread apart, and the foot brought down with a sudden stamp, very much the same but not so marked as that seen in locomotor ataxia. When the child tries to stand he sways from side to side and if the feet are approximated there is distinct oscillation of the body due to an attempt in the weakened muscles to preserve the balance. After the ataxia is well developed rigidity of the limbs due to an affection of the motor tract in the lateral columns becomes manifest. There is now added a spastic element to the gait with a tendency to drag the feet and muscular weakness. Even in this early stage of the disease an examination of the eyes will show a lateral oscillation of the eyeball on attempted movements; there is, however, no affection of the ocular muscles or the optic disk. In advanced cases the rigidity of the lower extremities becomes marked, the loss of power almost complete, the reflexes abolished, and the arms so ataxic that attempts at movement results in irregular, slow, choreic-like movements. There may be also some loss of power in the arms. Speech is now slow,

FIG. 182



Friedrich's hereditary ataxia. The awkward posture and forward bending of the body are noticeable; also the slight flexion of knees and elbows. (Start.)

indistinct, and difficult, with fibrillary tremors of the tongue. The sensation is usually normal and becomes affected only late in the disease. There is usually no pain and no disturbance of the function of the bladder or rectum. In the advanced cases the feet present a very characteristic deformity; they are apparently shortened and in a condition of *pes cavus*; the toes are hyperextended, and this is especially true of the great toe, which is drawn back like a hook. The mentality of those affected is usually defective; they are educated with difficulty, and remain in a backward or even infantile mental condition.

**Prognosis.**—Prognosis is favorable as to life and absolutely unfavorable as to cure. There is no known method of treatment which can appreciably affect the progressive course of the disease.

**Diagnosis.**—The only condition from which it is to be differentiated is a *cerebellar form of ataxia* described by Marie. This disease was first described by Marie and has the titubation, ataxia, tremor of the head and of the extremities, and the nystagmus seen in Friedreich's ataxia. It, however, differs in several essential particulars. Atrophy of the cerebellum has been found in three cases. The spinal cord was not diseased. Hereditary cerebellar ataxia, however, comes on after the age of puberty with some loss of power in the legs and a moderate ataxia, but not so marked as that of Friedreich's disease. There are marked disturbances of sensation. Amblyopia and contraction of the visual fields due to atrophy of the optic nerve may be present. Diplopia and color blindness have also been described. The extensive deformity of the foot and the kyphosis of Friedreich's ataxia are not present. This is also a family disease and several members of the same family may be affected.

**Treatment.**—The prognosis and treatment are as hopeless in the cerebellar form of ataxia as in Friedreich's ataxia.

While nothing can be done to cure either one of the above diseases, much may be done to prolong life and to make the patient comfortable. Attention to the body functions and especially to nutrition and to the gastroenteric tract, plenty of fresh air and sunshine, and mild massage associated with hydrotherapy to assist in keeping the muscular system in good condition, give better results than medicinal measures. In the later stages care should be taken to prevent contractures and deformity. Not only does section of the tendons relieve the deformity, but often overcomes the increased tension and by the resulting relaxation of the muscles relieves the discomfort or even pain due to spasm. Death occurs after years of invalidism from some intercurrent affection.

#### HEREDITARY SPASTIC PARALYSIS.

Hereditary Spastic Paralysis, or Family Spastic Paralysis, is a condition of spastic paralysis affecting the lower extremities, at times to a slight degree the upper extremities, and occurring as a family disease with heredity as an important factor. It may be due to different causes.

rtically all of these cases, however, depend on an abiotrophy and defective development of either the brain or of the spinal cord (Figs. 183, 184). We may therefore classify them into two distinct types: (a) Those due to a defective development of the motor tracts (crossed tracts of the spinal cord). (b) Those due to an arrested cerebral development.

(a) In the first group of cases there is no evidence of cerebral disease. There is simply an affection of the spinal motor tracts. These may

FIG. 183



Spastic paraplegia: walking or standing alone impossible. (Dercum.)

FIG. 184



Spastic paraplegia: crossed-legged progression. (Dercum.)

as isolated cases (Little's disease), in groups in an individual family without previous heredity, or there may be a history of cases in the immediate preceding generation or in collateral branches of the family. They may develop in early childhood or less frequently later in life. In a family which recently came under my observation, several members presented a paralysis, spastic in type, developing about puberty. This was progressive. This family represented a type of this disease. There is a development in late childhood of a loss of power associated with spasticity, a disturbance of the gait, and associated with increase of reflexes, the Babinski reflex, and ankle clonus. There is no loss of sensation, no disturbance of the bladder or rectum, and no true



ataxia. The mental condition of these children and those who have gone on to adult life is practically normal. There is no evidence in any of the cases that accidents at birth or acquired cerebral disease had aught to do with the production of the symptoms. The disease is a progressive one and finally results in such loss of power as to confine the patient to a rolling chair or to bed.

(b) *Arrested Cerebral Development. Amaurotic Family Idiocy.*—Cases of this kind, first described by Freud and Sachs, of New York, occasionally occur. Most of them are seen in Jewish families. A child who is born apparently healthy and of good physical and cranial development does well for several months to a year and then begins to show evidence of arrested cerebral development. The mental faculties either come to a standstill or retrogress and a condition of idiocy is presented. Nystagmus occurs and is associated with progressive blind-

FIG. 185



Little's disease. The spastic rigid condition of the muscles is shown when the child is at rest. The child was very bright and only presented the spastic condition of the muscles.

ness. This is due to an atrophy of the optic nerve and a grayish-white opacity in the region of the fovea centralis. This may be all that is presented. In other cases a spastic paraplegia of the lower extremities develops; there is tremor of the arms, due probably to loss of power; excessive slowness in whatever speech may be present, and finally death after one or two years from progressive emaciation. Convulsions are never present. The pathology of this condition consists in a complete arrest in the development of the cells in the cerebral cortex.

**Diagnosis.**—The diagnosis of the purely spinal type is comparatively easy. Given several members of the same family affected by spastic paralysis of the lower extremities without sensory derangement or disturbance of the bladder or rectum, there is no other condition with which it could be confounded with a possible exception of those rare cases in which several members of the same family present a cerebral form of paralysis due to the fact that in a contracted pelvis in the mother prolonged labor, or the application of forceps resulted in brain injury



in successive labors. These cases date from birth and are associated with convulsions, marked mental defect, as a rule, and the different individuals may present different types of paralysis. (See Cerebral Paralysis of Childhood, p. 957.)

Amaurotic family idiocy due to arrested cerebral development presents a clinical picture so distinctive and the history of its occurrence in several members of the same family that it could hardly be confounded with any other disease. A brain tumor in a child would give blindness with motor symptoms, but there would in all probability in these cases be convulsions or other localizing symptoms. The ophthalmoscopic examination would undoubtedly make the diagnosis as the optic atrophy and retinal changes of family idiocy are entirely unlike the choked disk of brain tumor.

**Prognosis.**—The prognosis in both class of disease is unfavorable.

**Treatment.**—There is no known treatment that effects the course of either.

#### PROGRESSIVE MUSCULAR DYSTROPHY.

The term Muscular Dystrophy will be used in this section to designate a group of diseases distinctly localized to the muscular structures in order to distinguish them from another class of diseases to which the term progressive muscular atrophy has been applied and due to disease of the anterior-horn cells of the spinal cord. It will therefore be understood that unless otherwise so stated the central and peripheral nervous systems present no pathological conditions. Progressive muscular dystrophy represents an abiotrophy of the muscular tissues. Any of the voluntary muscles of the body may be affected. Several types of the disease have been described, depending upon the distribution of the muscles affected. There is little necessity from a clinical or pathological standpoint of following these artificial types. The different types gradually merge one into another and there are cases which cannot be assigned to any particular group. The types described are as follows: (a) Landouzy-Dejerine type affecting the face and shoulder girdle. (b) Erb's type, the juvenile form of muscular dystrophy in which the muscles of the shoulder girdle, the pelvic girdle, and the back are affected. (c) The pseudohypertrophic form of muscular dystrophy. This refers more to the type of muscular change and secondary fat deposition than to the muscles involved.

**Etiology.**—This disease occurs in families and sometimes all of the members of a family are affected. There may be a true heredity, several generations being affected. More males than females show the disease. The transmission of the disease is usually through the mother. The infectious fevers and traumatism have been suggested as causative factors, but are probably accidental. The disease represents an inherent congenital defect of muscle vitality leading to degeneration in certain groups early in life. Members of collateral branches of families may be affected with perhaps only a sporadic case in one family.

**Pathology.**—The most careful examination of the brain and spinal cord even in those cases that have lasted many years (as in Spiller's

and Dejerine's cases) has shown no change in the central or peripheral nervous systems. The examination of the muscles shows a marked atrophy of the individual muscle fibres with an increase in the number of the muscle and interstitial nuclei. With this atrophy of the individual muscle fibres the striations are lost and finally when there is a marked atrophy of the muscle the muscle substance of some of the fibres disappear, leaving the sheath filled with an edematous exudate or its place is taken by a deposit of fat.

In some cases the interstitial connective tissue is larger in amount with an increase in the deposition of fat. Individual muscle fibres may be hypertrophied and large giant muscle cells and fibres following the appearance of normal fibres may be present. The proliferation of

FIG. 186



Pseudohypertrophic muscular dystrophy. Four brothers, aged twelve, eleven, eight, and seven years. The calves and the anterior surface of the thighs are hypertrophied. The muscles of the back are atrophied. The eldest has so much weakness of the muscles of the neck that he cannot hold up his head. (Curschmann, *Klin. Abbildungen*.)

connective tissue and the deposition of fat which are most marked in the pseudohypertrophic form are secondary to the atrophy of the muscle fibres. The giant muscle fibres are probably an attempt at compensatory hypertrophy.

**Symptomatology.** (a) *Landouzy-Dejerine Type.*—This type usually develops in early childhood, but I have seen two cases develop in adult life. The peculiar features are the early atrophy affecting the muscles of the face beginning in the orbicularis oris and extending to the levator menti, the risorii, and later to the other muscles of the face. The lips

become weak and cannot be firmly closed, the mouth is held open with protruded lips. The upper face muscles usually escape. As the disease progresses the muscles of the neck and shoulder girdle become affected. There are no fibrillary tremors of the muscles, no disturbance of sensation, and the reaction of the muscles to mechanical and electric stimuli is gradually lost. The tendon reflexes diminish with the loss of muscle power.

FIG. 187



Pseudohypertrophic paralysis. The act of rising. This position shows the weakness of the muscles of the neck and the atrophy of the arms. (Starr.)

FIG. 188



Pseudohypertrophic paralysis. The act of rising. (Starr.)

(b) *Erb's juvenile type* usually begins between the twelfth and sixteenth year, and in rare instances even later. The muscles of the shoulder girdle are first affected. The pectoral muscles, the trapezii,

FIG. 189



Pseudohypertrophic paralysis. The act of rising. (Starr.)

FIG. 190



Pseudohypertrophic paralysis. The act of rising. (Starr.)



PLATE XXVII.



Pseudohypertrophic Paralysis.



latissimus dorsi, rhomboids, and deltoids are successively affected. There may be a true atrophy or the muscles may maintain their original size or even be slightly increased in size, but with progressive loss of power. There is difficulty in elevation of the arms; the shoulders are thrown forward and the scapulae project away from the chest; and when the muscles of the back become involved lordosis appears. If there is no arrest of the disease the lower extremities become affected beginning in the muscles of the hip and progressing downward to the feet. The difficulty in walking due to loss of power in the lower extremities is accentuated by the affection of the back muscles, until finally the patient is confined to the wheeling chair or to bed.

(c) *Pseudohypertrophic Form*.—This form usually begins early in childhood between the second and seventh years with an increase in the size of the calves of the legs and of the thighs. With this increase in size there is a distinct loss of power with a clumsy and awkward gait (Fig. 186). Fatigue develops after slight exertion and accentuates the awkwardness and weakness of the lower extremities. When the disease is fairly well advanced the child experiences much difficulty in arising from a sitting or recumbent posture. It soon learns to use the hands to assist it and literally climbs up itself by pushing with the hands upward along the leg until it assumes an erect posture (Figs. 187 to 190). When the muscles of the back become affected the forward curvature of the spine of the lumbar region with a backward position of the shoulders and cervical spine to compensate for this gives a peculiar standing attitude (Fig. 191). The legs are held wide apart and the gait is described at this period as waddling. The muscles of the shoulder girdle and arms later become affected and present the same pseudohypertrophy (Fig. 192). The muscles of the forearms and hands if affected at all are only so in the very latest stages of the disease. There is no disturbance of sensation. A diminution of the reflexes occurs when the loss of power becomes marked. The electric reactions here are normal, but there is a progressive failure even to increased quantities of the galvanic current. The disease may become arrested, but is usually slowly progressive until in adult life some intercurrent affection causes death. (See Plate XXVII.)

**Prognosis.**—Prognosis as far as life is concerned is altogether favorable in all these

FIG. 191



Pseudohypertrophic paralysis. The calves are large; the back is weak and curved forward. Deltoids and triceps are atrophied, serrati are weak, hence the scapulae protrude. (Starr.)

classes of cases. In advanced cases the diaphragm may be involved and thus lead to pneumonia, tuberculosis, or other respiratory affection or the patient may die of respiratory paralysis. Arrested development occurs in a small number of cases.

**Treatment.**—The treatment of all forms of muscular dystrophy is practically the same. It is better for children to live as much as possible in the open air, preferably in the country. The nutrition should be

FIG. 192



Pseudohypertrophic paralysis; five years after onset. Muscles of arms and legs greatly hypertrophied. Both feet contracted and in a position of talipes. (Curschmann, *Klin. Abbild.*)

good and the exercise carefully regulated. Well-directed massage, passive and resistive movements offer the best methods of treatment. Fatigue either by exercise or by massage should be carefully avoided. Electricity carefully applied in moderate quantities, but not to produce contraction, is of value. In the cases where the trunk is early affected and where sufficient power for locomotion still remains, lower extremity light braces or plaster-of-Paris jackets are of



**Peroneal Type of Muscular Atrophy.** (Charcot-Marie-Tooth Disease.) This disease is the Progressive Neural Muscular Atrophy of Hoffman, and was long considered to be a form of muscular dystrophy affecting the lower extremities, but in 1889 Hoffman found changes in the peripheral nerves sufficient to account for the symptoms. It is therefore not a muscular dystrophy in the strict sense of the term, but a degenerative muscular condition secondary to changes in the nerves.

**Etiology.**—Heredity seems to be an important factor. It also occurs as a family disease. It may begin at a very early age or may not occur until as late as twenty. Exposure to cold and wet has been given as an etiological factor.

**Pathology.**—The pathological lesion described by Hoffman was an atrophic neuritis in the peroneal nerves and with degenerative changes in the muscles like those of muscular dystrophy (Fig. 193). The later investigations have also shown slight sclerotic changes in the posterior columns of the spinal cord and in the posterior spinal ganglia. It may, therefore, be considered to be an affection of the entire peripheral sensory path with a peripheral degenerative neuritis.

**Symptomatology.**—The lower extremities are at first affected. There is a progressive loss of power with atrophy beginning in the muscles of the feet and in the long peroneal muscles on the outer side of the leg. This is followed by an involvement of the tibialis anticus, extensor communis digitorum, and later of the calf muscles. This results after a few years in such complete loss of power as to incapacitate the sufferer from either standing or walking. The development of toe-drop and the weakness of the muscles about the knee give rise to a peculiar gait.

The foot is lifted high with the legs held wide apart, and as the foot is brought down the foot falls outward. The muscles are the seat of fibrillary contractions, and if examined by the galvanic current in the early stage full reactions of degeneration may be obtained. In later stages there is a progressive failure to react to either faradic or galvanic

FIG. 193



Charcot-Marie-Tooth disease. Peroneal atrophy. Atrophy of the legs and drop-feet, and atrophy of the hands. (Starr.)

current. The spindle-shaped appearance of the limb is due to the distribution of the atrophy. It is confined to areas below the knee, while the thigh muscles remain normal. In exceptional cases the thigh muscles may be affected and occasionally the small muscles of the hand, of the forearm, and arm may be later involved. In unfavorable cases there is progression in the late stages to the muscles of the trunk, with death from some intercurrent affection.

The reflexes in the affected areas are lost comparatively early in the disease. There may be loss of sensation or simple diminution along the outer side of the legs.

**Prognosis.**—Prognosis for life is good. Prognosis for recovery of function is unfavorable. Some cases come to a standstill after the affection has reached the knee. In other cases the disease extends no farther than the affection of the lower leg and the forearm. In a very small number of cases the paralysis may extend to practically all the voluntary muscles of the body.

**Treatment.**—The treatment should be that of a tonic, stimulative character with attention to the general health and the use of massage, graduated movements, electricity, and hydrotherapy.

#### MALFORMATION AND IMPERFECT DEVELOPMENT OF THE SPINAL CORD.

We shall not consider here those conditions, such as the entire absence of the spinal cord or of the posterior spinal ganglia, which are merely of a scientific interest and have no practical bearing.

**Spina Bifida.**—This is a frequent condition in infancy and childhood (Fig. 194). It is said to occur in one case out of every thousand births.

**Etiology.**—Inasmuch as the spinal arches in the lumbar cord are the last to close any interference with this process will predispose to the formation of the condition under consideration. The accumulation of fluid in the meningeal tumor is secondary to lack of resistance and does not depend either upon a congenital increase of fluid or increased secretion of cerebrospinal fluid.

**Pathology.**—The careful study of the spine of large numbers of children will reveal at times evidence of defective development of the spinal arches. In the simplest form it may be merely an absence of the spinous process of a single vertebra. In others there appears to be a diminution in size of the vertebrae and a failure of the laminae. In still others there is an entire absence of the bony structures closing the spinal canal either of a single or of several vertebrae. When several vertebrae are deficient a protrusion of the membranes of the cord filled by cerebrospinal fluid takes place. This condition is spoken of as *meningocele*. The wall of the sac is lined by the arachnoid but not always by the dura. This latter may be congenitally absent over the tumor. The entire structure is covered by the skin.

In the more complicated cases the lower portion of the spinal cord protrudes into this sac and with it the nerve roots (*meningomyelocele*). In still other cases the lower portion of the cord is extended to form a cavity and this is enclosed within the meningeal sac (*syringomyelocele*). A considerable accumulation of connective tissue and fat may be present (Fig. 195).

**Symptomatology.**—Simple meningocele without involvement of the spinal cord may be associated with very few symptoms. As a rule, however, there are other congenital defects, such as club-feet, ectopia of the bladder or other viscera, hydrocephalus, etc.

FIG. 194



Spina bifida.

When the spinal cord is included in the sac the motor and trophic actions may be disturbed. There may be spastic or flaccid paralysis with atrophy, anesthesia, disturbance of bladder or rectal function, depending upon the degree of involvement of the cord. The pressure from an increased amount of fluid may be a factor in the production of these symptoms.

The physical and mental development of the child is backward and poor; the nutrition weak and easily disturbed. The skin covering the fluctuating tumor may be perfectly normal or may be very thin, due to pressure.

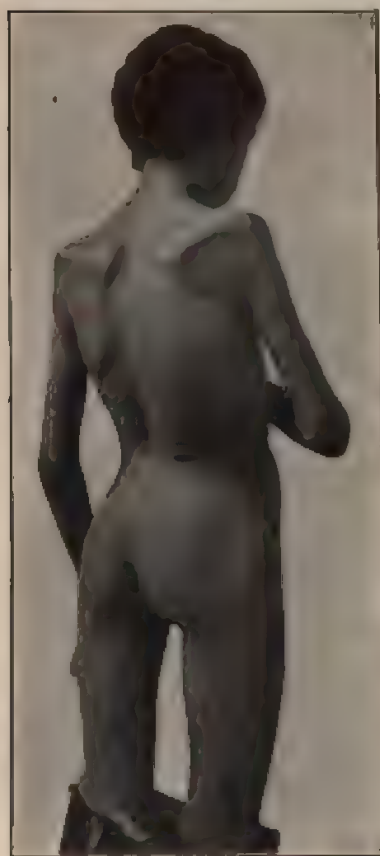


**Diagnosis.**—The diagnosis is usually easy. I have, however, seen a lipoma of soft consistence directly over the spine mistaken for a meningocele. The presence of a bony lamina beneath and the absence of true fluctuation made the diagnosis. In doubtful cases hypodermic puncture and the presence of cerebrospinal fluid with its distinctive microscopic and chemical qualities will separate the cyst of a spina bifida

from that due to other conditions. The x-rays may also be used to determine the absence of bone.

**Prognosis.**—Not infrequently the sac ruptures during or shortly after birth, with a fatal result. Septic infection from ulceration of the skin overlying the tumor with the production of meningitis is very likely to occur. In simple meningocele without involvement of the spinal tissues prognosis is altogether favorable as far as life is concerned. This area, however, must always be carefully protected to prevent septic infection. In meningomyelocele and syringomyelocele prognosis is unfavorable.

**Treatment.**—Apart from the general care of the child's health and the protection of the tumor from injury the treatment is entirely surgical. The surgical procedure depends on the morphology of the tumor. In some cases a meningocele represents a true type of sacculated hernia of the meninges in which a large sac communicates by a small opening with the spinal canal. In such cases a ligature may be applied and the sac cut away. In other cases where there is free communication, more extensive surgical procedures with closure of the spinal canal may be attempted. In any event the operation is a serious one and should not



Case of syringomyelia. Atrophy of the muscles of the shoulders and right arm. Curvature of the spine forward from atrophy of muscles of the back. (Starr.)

be undertaken where there is extensive hydrocephalus or evidence of complete destruction of cord function. Operation should not be attempted until some time after birth (at least several months) and only when the physical condition of the child warrants such a serious procedure.



## CHAPTER XXXVII.

### DISEASES OF THE BRAIN AND MENINGES.

#### MENINGITIS.

INFLAMMATIONS of the membranes of the brain may be acute, sub-acute, or chronic. They may be localized to the brain alone, the spinal cord, or may affect both. Meningeal affections are especially frequent in infancy and there is a special form described by Gee and Barlow to which the term non-tuberculous leptomeningitis infantum has been given. Koplik and others have considered many of these cases to be infantile types of epidemic cerebrospinal meningitis.

**Etiology.**—In the majority of cases inflammation may be traced to an infection of the meninges by some pathogenic organism. It most frequently occurs in association with the acute infectious fevers. Pneumonia, erysipelas, septicemia, and tuberculosis are the most frequent causes. It is, however, found in association with or following typhoid fever, smallpox, scarlet fever, measles, diphtheria, influenza, and rarely with rheumatism and mumps. A chronic inflammatory condition of the meninges is met with in tuberculosis and syphilis. Traumatism with or without an involvement of the meninges may be followed by meningitis. It occurs by extension from neighboring inflammatory processes and especially from mastoid disease and disease of the middle ear. Inflammatory processes in the nose may extend to the brain. Infection may also take place from suppurative conditions of the sinuses or may be transmitted to the meninges by operative procedures on the sinuses. Septic emboli or blood infections from pus accumulations elsewhere in the body and especially those due to bone disease not infrequently infect the meninges. A special form of meningitis, the epidemic cerebrospinal meningitis, is due to the diplococcus intracellularis of Weichselbaum. A study of the causes of meningitis will reveal that the disease is due either to the infection of the cerebral membranes by the specific organism causing the disease with which it is associated or to a mixed infection with some septic process such as occurs in tuberculosis. We may therefore have in such cases a type of inflammation characteristic of the causative agent, such as tuberculous meningitis, entirely different from that due to the mixed infection—*i. e.*, the septic meningitis. The most common organism found in the septic type of meningitis is a streptococcus or a staphylococcus, although any of the pyogenic organisms may be found.

**Pathology.**—In acute meningitis the inflammation is localized to the soft membranes of the brain and especially to the pia mater in direct

contact with the brain substance. While the pia mater covering the entire brain is usually more or less involved there is a tendency in certain forms of meningitis to a localization in certain definite areas. Thus in the septic processes extending from middle-ear disease the inflammation may be found only on one side of the brain or even localized to a small area. In septic meningitis in the great majority of cases, whether primary or secondary to other processes or infections, the convexity of the brain is more likely to be infected than the base; whereas in the epidemic form, while the entire brain and spinal cord may be affected, the part most involved, as a rule, is the base. In the meningitis of infancy described by Gee and Barlow the process is localized to the posterior portion of the base of the brain. Tuberculous meningitis elects the base of the brain, and especially the anterior part near the optic commissure.

Inflammatory conditions of the meninges do not differ in their pathology from that seen in other serous membranes. We may divide the pathological manifestations into three stages: (1) a stage of congestion, (2) a stage of effusion, and (3) a chronic adhesive stage. The effusion may be either serous in type or if the infecting agent be a pyogenic organism there is an accumulation of pus.

*Stage of Congestion.*—There is a marked hyperemia affecting the pia mater shortly followed by an exudation of serum, lymph, fibrin, and a few leukocytes. The surface of the brain appears a bright red, and on close examination the membranes have lost their smooth, shining appearance and are dull and roughened. In passing the finger lightly over the inflamed areas a decidedly roughened, adhesive feeling will be imparted. The microscopic examination at this stage shows a marked distention of the capillaries, an extravasation of red corpuscles here and there, a film of fibrin on the surface of the brain, and an accumulation of small round cells around the bloodvessels and free in the meshes of the pia and arachnoid. This process is not confined to the meninges, but may be followed in marked cases by a similar change along the bloodvessels, extending from the pia into the brain cortex. The inflammatory process may stop at this congestive stage with little damage to the cerebral tissues. When the meningitis affects the base, however, even a dry meningitis of this grade may cause serious damage to the cranial nerves. This is especially true of the nerves of special sense, the auditory and optic nerves. In cases where the entire pia mater is involved the same changes are found in the extension of the pia into the brain ventricles—i. e., in the choroid plexus.

*Stage of Effusion.*—In inflammatory conditions at the base of the brain obstruction of the communication between the ventricles and the subarachnoid space by the inflammatory exudate may occur and two closed sacs are formed; one an internal closed area comprising the ventricles, the other the subarachnoid spaces. Even when obstruction does not take place the accumulation of fluid may be very extensive and cause serious pressure on the brain. In septic processes the convexity

and base of the brain may be bathed in pus. In those cases where the ventricles are closed the accumulation of fluid produces a condition of internal hydrocephalus. The ventricles are markedly distended and in the case of infants the pressure is sufficiently great to cause a separation of the sutures and produce a globular enlargement of the head.

A purulent process of the meninges extends along the bloodvessels into the brain substance, and is followed either by an inflammation of the cortex by extension or small abscesses.

*Chronic Adhesive Stage.*—In cases of intense inflammation in which the plastic exudate is extensive adhesions between the membranes of the brain may occur. They are more frequently seen at the base of the brain and by involvement of the cranial nerves produce serious sequelæ. The organization and contraction of the exudate give rise to such pressure upon the cranial nerves as to cause partial or complete degeneration. The previous inflammatory condition of the nerve tissues by extension from the meningeal involvement is also a marked factor in this degeneration of the cranial nerves following meningitis. If complete obstruction of the aqueduct of Sylvius or the foramina at the base of the brain connecting the ventricles with the subarachnoid spaces takes place, either from the inflammatory process or as a result of secondary adhesions, a chronic hydrocephalus is produced. (See p. 967.)

*Symptomatology.*—The symptoms of meningitis will depend upon the intensity of the septic process and the age at which the child is affected. In infants the symptoms develop suddenly with high fever, although the temperature in some cases may vary only between 100° and 101° F. The early symptoms will be those due to the intense congestion and inflammation of the first stage. As early as the second day there is evidence of marked pain, referred to the head; there may be tenderness over the scalp, marked irritability, and disturbed sleep. The child buries its head in the pillow; there is retraction of the head; stiffness of the muscles at the back of the neck; photophobia, and increased sensitiveness to even slight sound. Vomiting may occur early, and usually by the third or fourth day the restlessness and twitchings of the muscles are followed by convulsions. The symptoms may subside here without loss of consciousness, or more frequently go on to the second stage, when evidence of hydrocephalus occurs and death supervenes. A case following this type will run its course in from six to eight days.

Besides this septic type of meningitis with a rapid involvement of the entire meninges, there is a class of cases in which the intensity of the infection and the resulting pathological process is much less marked than that above described. Here again the disease may come on suddenly with a chill or even a convulsion, but the fever is not so high and the disease runs a much longer and milder course. The process is not general, but localized to some one portion of the brain. In one of the cases reported by Gee and Barlow the process was so slight as to result only in a congestion, with dulling of the lustre of the meninges.

There may be even in some cases running a mild course distinct pus formation. When the meningitis affects the vertex (the convexity of the brain) the manifestations are entirely different from those in which the base is affected. The anterior part of the convexity is more likely to be affected than the posterior. The symptoms produced may be very mild, and easily overlooked on account of association with the infectious fevers. There is some headache and vomiting. Retraction of the head, if it occurs at all, is only slight and has to be carefully looked for. Convulsions sometimes occur and may be partial and localized to one or more parts of the body, but may later become general. General epileptiform convulsions may be very severe, frequently repeated, and associated with high temperature. The spasms are usually clonic in type and lead to marked exhaustion. There is, as a rule, no tonic contracture of the extremities, and while Kernig's sign is usually present it may in rare cases be absent. There may be an associated inflammation of the meninges surrounding the spinal cord. There will then be evidence of pain and tenderness along the spine, hyperesthesia of the skin of the trunk and of the extremities, individual muscular twitchings, and varying paralyses affecting one or all of the extremities.

**Posterior Basic Meningitis.**—This is a localized meningitis of the base of the brain. The primary seat of the inflammation is in the area of junction of the brain and spinal cord where the cerebellum overlaps the medulla. From here it extends forward along the transverse fissure into the ventricles or along the base of the brain as far as the optic commissure, involving the inferior surface of the temporosphenoidal lobes, and may extend downward along the upper portion of the spinal cord. The convexity of the brain is usually not affected, or at the most only very slightly. While the process is usually of a plastic, fibrinous character it may go on in severe cases to suppuration. In cases that recover chronic adhesions may unite the cerebellum to the medulla, obstruct the communication of the ventricles with the subarachnoid space, or, by closing the aqueduct of Sylvius, lead to chronic hydrocephalus. The accumulation found at autopsy in the ventricles of these cases is opaque, due to large flakes of inflammatory lymph, or even pus, with high specific gravity—in other words, of inflammatory origin. Koplik and others have recently reported the finding of the diplococcus of epidemic cerebrospinal meningitis in some cases of this type of meningitis occurring during an epidemic. The diplococci were present both in the cerebrospinal fluid and in the exudate. There is no doubt that a certain group of cases of epidemic cerebrospinal meningitis in infancy presents the clinical picture of posterior basic meningitis. I do not believe that all cases of posterior basic meningitis are due to the diplococcus of Weichselbaum. The clinical type is sufficiently distinct to warrant separate consideration.

**Symptomatology.**—The symptoms develop suddenly in the mild cases and run a long course of from four to six weeks; the temperature is not very high, ranging, as a rule, from 98° to 102° F.; in some cases it may not be above 100°, or at the most 101° F. But even in the subacute mild



ases terminal hyperpyrexia even as high as 107° or 108° F. may occur; in other cases a subnormal temperature develops as death approaches. The most important and persistent symptom is retraction of the head; the rigidity of the neck muscles may go on to opisthotonos. Vomiting is frequent and may be the first symptom. Nystagmus due to cerebellar cortical irritation is frequent; strabismus is common. The pupils in the early stage are contracted and later become irregular and markedly dilated. Examination of the eye-grounds reveals optic neuritis only in a small number of cases, but this in itself is of value in differentiating this form of meningitis from tuberculous meningitis, which also affects the base and is of much more frequent occurrence. Blindness, transient in character, may occur without changes in the optic nerve and is probably due to interference with the optic paths in the neighborhood of the optic thalamus. In the early stages there is considerable restlessness and irritation, followed in severe cases by torpor and coma. Convulsions are rare, but it is not infrequent to find in severe cases a marked condition of persistent tonic spasm of the extremities. In such cases the clinical picture is typical; the head is retracted, the arms are extended and rigid, the hands flexed and held outward, and the lower extremities in marked extension. The spasm in some cases may be flexor in type, with the body bent forward, the head in extreme retraction, and the legs and arms fixed in a semiflexed position. If the child be conscious attempts to reduce this spasm by changing the position of the limbs or reducing the retraction of the head leads to considerable pain and irritation.

**Diagnosis.**—The ease with which a diagnosis is made in a case of meningitis will depend mainly upon the time at which the case is seen. In well-developed cases the diagnosis is comparatively easy. In mild cases there is more difficulty. In the former the presence of a source of infection, with fever, somnolence, coma, inequality of the pupils, strabismus, nystagmus, and retraction of the head, gives a distinct clinical picture. The greatest difficulty in diagnosis is met with in rather a large class of cases in which symptoms very closely resembling meningitis are due to toxic irritation of the central nervous system. The French clinicians have enabled us to disguise our ignorance of the real nature of this affection under the term *meningismus*. At the beginning, during the course of or in convalescence from various infections, especially pneumonia, influenza, typhoid, the summer diarrheas, reflexly from dentition or from the retained toxins due to poorly drained intestinal suppurations, a series of symptoms develop which resemble very closely those present in meningitis and are often identical with them.

It may be stated that in some cases a clinical picture is presented that cannot be differentiated from simple meningitis and yet at autopsy no trace of an inflammatory condition of the meninges can be found. This is especially true in the forms of *meningismus* observed during the course of the intestinal infections. In older children where the Babinski reflex is of value—*i. e.*, where the normal plantar reflex is extension—extension of the toes to plantar irritation and the presence of Kernig's symptom will assist in differentiating the true from the false

form of meningitis. In pneumonia the cerebral manifestations may occur early, before the lung condition can be localized. The convulsions, the delirium, and the agitation diminish as the pulmonary symptoms increase and the evidence of an increase in the inflammatory symptoms which would cause a paralytic condition of the cranial nerves and the extremities does not occur. The variations in the symptoms following closely those due to the pulmonary disease should also point to a spurious form of meningitis. In the lobar type of pneumonia the cerebral symptoms diminish and rapidly disappear after the crisis. In typhoid the cerebral symptoms coincide with the intensity of the typhoid intoxication and here again disappear as this condition improves. In dentition and reflex disturbances due to intestinal parasites the removal of the causative factor is followed by a disappearance of the symptoms. The absence of fever and reflex conditions is an important aid in diagnosis. In older children the presence of hysteria must be taken into consideration in diagnosis, but the absence of fever, the variation of the course of the disease, the bizarre character of the convulsions, the typical disturbance of sensation, and the control of the symptoms by suggestion, will easily differentiate the two conditions.

Retraction of the head, intense headache, vomiting, vertigo, convulsions, and optic neuritis may be present in cases of *brain tumor* affecting the cerebellum and may be mistaken for meningitis. The development, however, is slow and progressive; there is an absence of fever and of knee-jerks, a marked ataxia of the gait, and the optic neuritis is much more marked and present in a much larger number of cases than in meningitis. In *meningeal hemorrhage* the onset is sudden with little fever, and the convulsions are very violent. The following case is an example of meningeal symptoms (meningismus) due to retained pus.

M. L., aged six years, suffering from a suppurating hip-joint disease developed fever varying from  $101^{\circ}$  to  $103^{\circ}$  F., with loss of appetite, restlessness, rigidity of the muscles of the neck, and marked retraction of the head. There was no paralysis nor convulsions; the child was very sick and grew rapidly worse. The dressings which had been applied by the nurse were negligently and irregularly done. A diagnosis of meningitis was made by the surgeon in attendance, but the slow onset, the absence of paralytic symptoms, the absence of convulsions, flexion of the toes to plantar irritation, the absence of Kernig's symptom led to a diagnosis of meningismus from retained pus. A thorough cleansing of the sinuses about the hip-joint, with proper attention to the frequent renewal of dressings, resulted in a rapid disappearance of the symptoms in two or three days.

The differential diagnosis from the epidemic form of cerebrospinal meningitis and from tuberculous meningitis will be considered under those headings. This diagnosis rests mainly on the examination of the cerebrospinal fluid obtained by lumbar puncture and in all cases of doubtful diagnosis positive results as to the cause of the disease may be obtained by a microscopic and bacteriologic study of the cerebrospinal fluid. (See p. 382.)

**Prognosis.**—The duration of cases of posterior meningitis is, as a rule, much longer even in fatal cases than either of the forms above described. The usual duration is from six to eight weeks, although minor symptoms with slight fever in favorable cases may persist for two or even three months. Hydrocephalus is a very frequent sequel. Deafness and blindness are not of such frequent occurrence in cases which recover from this form as in other forms of meningitis. The prognosis is also more favorable, which is, however, saying but little for the hope of recovery in the large majority of cases.

The clinical picture of all the above forms of meningitis varies with the period of life in which it develops. In late childhood going on to adult life the clinical picture approaches that seen in the adult. The onset is not, as a rule, so sudden and there may be prodromal symptoms of headache, malaise, irritability, etc. When the headache becomes more intense fever develops, the pulse becomes slowed; there is marked constipation, irritability to light and sound impressions, and evidence of irritation of the motor cortex. Slowness of the pulse and the constipation are not met with in infants, and when they occur in childhood are valuable early signs. After several days stupor develops and is followed at varying times in different cases by coma. The convulsions of the early stage are followed by a paretic or paralytic condition of the extremities. Trismus and grinding of the teeth are followed by dropping the jaw. The eye symptoms do not differ from those above detailed in the early forms. In the later stages the pulse becomes very rapid, the fever becomes higher, and death occurs. In the growing child and in the adult the pressure symptoms in the stage of hydrocephalus are more marked than in the infant on account of the unyielding character of the skull. The prognosis as adult life is approached is somewhat better than in childhood.

**Treatment.**—The treatment of meningitis is mainly symptomatic. Any possible source of infection, such as suppuration of the middle ear or of the mastoid or accumulations of pus elsewhere in the body, should be carefully treated. In infectious fevers in which meningitis is a frequent complication, the ear and the nasal cavities should be kept as clean as possible and where skin lesions are present they should also receive careful attention. The child should be placed in a quiet room and protected from all possible sources of irritation. If the temperature is high it should be controlled by hydrotherapy. An ice-cap to the head will assist in controlling the pain; a cold pack will very often relieve the agitation and irritability and assist in reducing the temperature. If constipation be present or even without this, calomel in divided doses has a beneficial effect early in the disease. This may be followed by an occasional saline purge later in the disease, with the idea of decreasing the congestion. Lumbar puncture may give in some cases considerable cloudy fluid with a diminution of pressure symptoms and as a therapeutic measure it occasionally gives excellent results; more often, and especially where the communication between the ventricles and the subarachnoid space is blocked by plastic lymph



or adhesions, the symptoms of pressure persist in spite of the operation. Operative procedures, such as trephining and drainage, drainage of the fourth ventricle after trephining the occipital bone, tapping the lateral ventricles through the anterior fontanel, etc., have all been attempted in a very small number of cases, but the results are unsatisfactory on account of the failure of children to react from the shock of the operation. They cannot therefore be recommended as routine procedures in this disease.

Lees and Barlow speak highly of the effect of paracentesis of the tympanic membranes where any suspicion of ear disease is present. They have also secured results from the use of potassium iodide in doses of 0.065 to 0.195 gm. (1 to 3 gr.) every two hours even in infants. Mercury in the form of mercury and chalk, 0.065 gm. (1 gr.) three times a day or by the inunction of 1.95 gm. ( $\frac{1}{2}$  dr.) of mercurial ointment daily, may be used. Excepting cases where there is distinct evidence of syphilis I have seen no results from the use of these drugs. Cases which recover are usually treated by careful attention to the general nutrition and to meeting individual symptoms by simple measures.

The tuberculous form of meningitis and the epidemic cerebrospinal type are to be found in their respective sections.

### ENCEPHALITIS.

Encephalitis is an inflammation of the cerebral tissues non-purulent in character. Acute localized inflammation of the cerebral tissues occurs more frequently in childhood than at any other time of life. This is undoubtedly due to the influences of the acute infections acting as predisposing factors.

**Etiology.** Encephalitis occurs during the course of or as a sequel to the following acute infections: influenza, scarlet fever, measles, diphtheria, pertussis, pneumonia, mumps, erysipelas, ulcerative endocarditis and other septicemic conditions. Ptomaine poisoning (from both fish and meat) and carbon dioxide intoxication are also factors.

**Pathology.**—Acute non-suppurative inflammations have been described in the preceding pages: in the peripheral nerves, as localized parenchymatous and interstitial neuritis, in the gray matter of the anterior horns of the spinal cord, in infantile spinal paralysis, and in the posterior spinal ganglia in herpes zoster. The pathological process does not differ essentially in any of the three conditions. There is a central zone of intense congestion with hemorrhagic extravasation, perivascular round-cell accumulation, and destruction of nerve tissue. About this area there is a zone of congestion. Secondary degeneration in the nerve elements affected follows and its distribution depends on the areas involved. When the acute inflammation subsides ependymal tissue is found in the area of destruction of nerve tissue. While any part of the encephalon may be affected there are certain areas of election. In the cortex the motor area is most frequently involved. The



gray matter surrounding the aqueduct of Sylvius is the seat of that form called the polioencephalitis superior of Wernicke. The cells of the motor nuclei of the cranial nerves are much more frequently affected than the sensory nuclei. I have seen cases where all the motor nuclei were affected without derangement of sensation. The cerebellum may also be involved.

**Symptomatology.**—The onset is sudden in association with one of the above-mentioned etiological factors. There may be in some cases a day or two of prodromal symptoms: depression, irritability, restlessness, etc. The period of active symptoms is ushered in by a chill or convulsion. If the area of inflammation is extensive this is rapidly followed by stupor and coma. In localized areas in the cortex both of these symptoms may be absent. The temperature is elevated and may rise as high as 104° F., but usually subsides after several days, falling slowly to normal. The pulse is rapid and may be extremely so. The respirations are usually regular, but may be hurried and in severe cases may approach the Cheyne-Stokes type. After the first or second day focal symptoms depending on the part of the brain involved develop. If the cortex is involved, a monoplegia affecting the arm or leg, a hemiplegia, or a paralysis localized to the lower portion of the face may be presented. In other cases there may be a cerebellar gait with nystagmus, etc. When the gray matter around the aqueduct of Sylvius is affected there is a partial or complete paralysis of the eye muscles. When the seat of inflammation is in the pons or medulla any one of the nuclei of the cranial nerves, several of them or practically all of them, may be involved, with paralysis of function in their distribution. In the severe cases the patient may never regain consciousness, dying in the period of coma. In other cases a period of excitability or even mania may follow for several days or weeks. In still other cases, and this is especially true of young children, imbecility as a result of deaf-mutism and destruction of the posterior portion of the brain on the left side follows. Where the disease process is limited there is, as a rule, very extensive improvement following the subsidence of the fever and the acute symptoms. It is rare to have complete recovery of function. A permanent paralysis, more or less extensive, remains.

**Diagnosis.**—This is always difficult on account of the resemblance to meningitis. The sudden onset with localization of the functional disturbance to one definite part of the brain to the exclusion of the rest of the brain mass, the absence of headache, retraction of the head and Kernig's sign, the rapid disappearance of the general symptoms, and the persistent leukocytosis should differentiate encephalitis from meningitis.

**Treatment.**—The treatment of encephalitis during the acute attack is absolute rest in a quiet room, a liquid diet, free purgation, and counter-irritation to the nape of the neck or over the scalp. This may best be obtained by blisters or the application of leeches. The treatment of the paralysis does not differ from that described under Poliomyelitis.

**CEREBRAL SINUS THROMBOSIS.**

Thrombosis affecting the sinuses of the dura mater of the brain may be local and confined to one sinus or may be extensive and affect several. A local thrombosis in the lateral sinus secondary to suppurative conditions of the middle ear and of the mastoid is most common. When it is extensive and widespread through many of the venous channels of the brain it is secondary to some general process. We may therefore divide the causes of sinus thrombosis into local and general systemic causes. Among the local causes the suppurative conditions of the middle ear and of the mastoid are the most common in childhood and in adult life, less often the cause in infancy. In infancy middle-ear disease most frequently gives rise to meningitis. Among the other local causes a phlebitis of the ophthalmic vein is most often secondary to phlegmons of the orbit, of the eye itself, or of the cavities of the face.

The thrombus formed in this vein may extend to the cavernous sinus and later to the other sinuses. Suppurative lesions of the nasopharynx and of the deep lymph nodes of the neck may also produce intracranial phlebitis. The suppurative lesions of the scalp due to traumatism, erysipelas, or anthrax may produce a venous infection carried by the emissary veins. Ostitis of the bones of the skull due to traumatism, tuberculosis, or syphilis is not an infrequent local factor. Among the general systemic causes septicemic and pyemic conditions running a subacute or chronic course and associated with a state of low vitality are most frequently to be found. The systemic causes are those usually found in widespread sinus thrombosis of infancy. Among these diseases may be mentioned the severe forms of gastroenteritis, cholera infantum, bronchopneumonia, tuberculosis, inherited syphilis, and following acute conditions such as scarlet fever, typhoid, and influenza. Middle-ear disease may produce very extensive sinus thrombosis through a general septicemia independent of the local irritative septic process. Bacteriological investigation has shown the presence of streptococci, streptobacilli, and the bacilli coli communi. It would seem that some infection was necessary, because the experimental occlusion of a cerebral sinus or even several of them is not followed by thrombosis (Ferrari).

**Pathology.**—The examination of the brain in children dying from thrombosis reveals a hard clot in the sinus and often an area of red, hemorrhagic, infiltrating extravasation in the area of the cerebral veins emptying into the affected sinus. Extensive or capillary hemorrhage may be present in the meningeal cavities and a clear or bloody fluid or gelatinous exudate may fill the ventricles. Section of the brain in the dark, hemorrhagic areas shows a capillary venous thrombosis with leaking out of the blood into the cerebral tissues. Gross hemorrhages are rare. In the infectious processes a cerebral abscess may follow if life is sufficiently prolonged.

**Symptomatology.**—The onset is usually sudden with evidence of cerebral or meningeal irritation. Coma rapidly supervenes; vomiting, convulsions, headache, and rigidity of the muscles of the neck may be present. In the local septic processes there is fever, whereas in the general septic conditions the temperature may be subnormal. The localizing evidences of the thrombotic process varies to a certain extent with the site of the thrombosis. When the lateral sinus is affected the veins on the affected side of the face and neck may not be present on inspection, whereas on the sound side they have a normal distended appearance. Local edema may be found in the mastoid area and may extend to the neck region. A hard, fibrous cord may be felt in the place of the jugular vein, due to the extension of the thrombus to the veins of the neck. The neck is sensitive to pressure, the muscles are rigid, and there may be some enlargement of the cervical lymph nodes. Paralysis of the facial nerve and deafness may be present, due to the local process in the ear.

Thrombosis of the superior longitudinal sinus secondary to local processes in the nasal cavities, the frontal or ethmoidal sinuses, or to a general septicemic process, is associated with cyanosis of the face, dilatation of the veins of the forehead and face, and depression of the fontanel. The fontanel may become prominent from an associated hydrocephalus. Profuse perspiration of the head and neck and epistaxis result from passive congestion in the areas drained by the superior longitudinal sinus.

Thrombosis of the cavernous sinus is associated with a slight exophthalmos of the affected side, amblyopia, paralysis of muscles of the eye, marked congestion of the veins of the retina, edema and swelling of the optic disk, and a cyanotic or red edema of the upper eyelid and of the forehead. As the thrombotic process progresses the other eye may also become affected, and these symptoms may be associated with those above described, due to the extension of the process into the other sinuses. When hydrocephalus develops there is coma, with vomiting, protrusion of the fontanel, the hydrocephalic cry, and convulsions. A purulent process may develop at any time either in the brain or in the local sinuses, and its occurrence depends on the vitality of the child and the pathogenic intensity of the infecting agent.

**Diagnosis.**—The diagnosis from meningitis is in most cases impossible. This is especially true in suppurative conditions of the middle ear and the mastoid. The presence of the edema about the mastoid and the neck, and a clot extending into the jugular vein, will often lead to the correct diagnosis. In extensive sinus thrombosis the marked cyanosis of the face, the epistaxis, the examination of the eye-grounds, the low condition of the vitality of the patient, and the weakened heart action could lead to a presumptive diagnosis. Abscess of the brain is not frequently a direct result of the thrombotic process, but the differential diagnosis is of no great importance, because in both cases an operation is demanded which will in itself reveal the exact stage of the process. Abscess of the brain from middle-ear disease may affect

either the temporosphenoidal lobe or the cerebellum. In the former case there would be word deafness—*i. e.*, inability of the child to react to spoken commands, but reaction to gestures, such as sticking out of the tongue after the examiner, etc. In abscess of the cerebellum there may be nystagmus, clumsiness of the same side of the body, and a forced position, the child always lying on the same side and returning to it if disturbed. The presence of high, irregular fever, with irregular chills and sweats, and a high leukocytosis with or without the above symptoms should lead to a diagnosis of abscess. Abscess may be present, however, with a normal or subnormal temperature. (*Vide infra.*)

**Prognosis.**—Thrombosis of the cerebral sinuses is a rapidly fatal affection in early childhood; death usually supervenes in a few days. In rare cases, and especially in later childhood, the duration may be longer and may even extend into weeks. If the irritative process be promptly removed a local thrombosis may in rare cases go on to resolution, with channelling of the thrombosis or complete obliteration of the sinus and recovery. In such cases, however, a sclerosis of the brain or hydrocephalus remains.

**Treatment.**—In extensive thrombosis of septicemic origin the treatment can only be palliative, such as is that employed in meningitis. An attempt should be made in all cases to control as far as possible the source of infection, and to increase the nutrition of the child by over-feeding and stimulation. The treatment of local thrombosis is surgical. A complete removal and cleansing of the local septic process, with removal of the clot from the sinus, and control of the hemorrhage by tampons of iodoform gauze is indicated. Care should be taken to exclude the possibility of an abscess of the brain before the wound is closed. Surgical treatment is now much better understood than formerly and an operation may save the life of a child.

#### ABSCESS OF THE BRAIN.

Abscess of the Brain, a comparatively rare condition in the adult, is of much more frequent occurrence in childhood on account of the frequency of purulent conditions of the middle ear and of the mastoid.

**Etiology.**—It may be stated as a general rule that a pyogenic infection is necessary for the production of an abscess within the cranial cavity. The so-called idiopathic or primary brain abscess is merely a confession of ignorance as to the source of infection. A localized abscess following traumatism or infection may be walled off and remain latent for years, to reappear with marked symptoms after a slight or extensive injury to the skull; this is the explanation of many of the so-called idiopathic abscesses. I have seen two abscesses the size of hazelnuts, one in either hemisphere of the brain, walled off from the rest of the brain by an old inflammatory capsule, and producing neither general nor localizing symptoms, and discovered by accident at the



autopsy. This condition is, however, of much less frequent occurrence in childhood than in later life, and even here it occurs infrequently. Septic infection from structures in direct relation to the intracranial cavity is by far the most usual cause of brain abscess in childhood. Purulent processes in the mastoid area and middle ear in later childhood may give rise to abscess of the brain, extradural abscess, thrombosis of the sinus, or acute meningitis. In many cases one or more of these may be combined. When the extension of the process is direct the dura is first becomes involved, followed by a localized or general involvement of the pia and arachnoid, and later of abscess within the brain. The infection may occur through lymphatic channels, and an abscess of the brain substance may be present without involvement of the meninges. When the infection extends from the roof of the mastoid cavity the abscess is usually found above the tentorium, and localized either in the sphenoidal lobe or posterior to this in the occipital area. When the infection extends from the posterior wall the cerebellum is usually involved, with or without thrombosis of the lateral sinus. When the infection is due to extension from the nasal cavities, the frontal or ethmoidal sinuses, the abscess is usually found in the frontal lobe, with or without meningitis of the anterior fossæ of the skull.

Next in frequency to the above causes traumatism is the most important factor in childhood. The traumatic infection may occur with or without lesions of the superficial tissues. Localized abscesses of the scalp, fracture or necrosis of the cranial bones, and punctured wounds are among the causes found. Purulent meningitis, a local abscess beneath the dura, or an abscess within the brain without meningeal involvement may be so produced.

Cerebral abscesses as a result of a general pyemic condition may be found scattered throughout the brain, but are of such infrequent occurrence in childhood as to demand little consideration. A septic infection of an extensive sinus thrombosis may result in extensive and multiple abscess formation.

**Pathology.**—In the great majority of cases the abscess is solitary and infiltrating. It is much more extensive, as a rule, when the cerebrum is affected than when it is situated below the tentorium. The abscess cavity varies greatly in size, in some cases being so small as to escape careful exploration in its immediate neighborhood during an operation. Smaller abscesses are, as a rule, fairly well separated from the unaffected brain substance by an inflammatory wall, and the blunt edge of a grooved director may easily pass over it without penetrating the abscess cavity. In other cases the abscess may destroy a large part of the cerebral hemisphere; there is no distinct wall; the brain substance in the immediate neighborhood is very edematous, and microscopic section shows marked round-cell infiltration ending into normal tissue. Encapsulation of an abscess may be very well developed, and yet a secondary extensive abscess may form in its immediate neighborhood. The examination of the contents of the abscess cavity shows a greenish-yellow or reddish-brown fetid pus,

containing leukocytes, pus cells, destroyed brain tissue, and infecting micro-organisms. Streptococci, staphylococci, pneumococci, and the bacillus pyocyaneus have been found in the pus.

**Symptomatology.**—The symptoms of brain abscess in children vary greatly with the intensity of the process and the presence or absence of complicating lesions of the meninges or of the cerebral sinuses. The symptoms may develop rapidly after operations on the middle ear or on the mastoid, but otherwise are, as a rule, of slow onset, but running a rapid course after the complete formation of the suppurating process. In a child who has chronic ear disease, persistent headache, irritability, and mental dulness may be the first evidence of intracranial involvement. The headache comes in paroxysms, and may occasionally be associated with vomiting. After several days or a week, or in some cases several weeks, the symptoms are markedly accentuated, the headaches become constant, the patient becomes anemic and sallow, there is a slight rise of temperature with marked loss of mental power, mental and physical fatigue, coating of the tongue, nausea, and vomiting. When this stage is reached the disease runs a rapid course. The temperature now drops to normal or subnormal, unconsciousness develops, the pupils become unequal, optic neuritis may be present, and there is often paralysis of the opposite side of the body. The pulse may remain normal, but is usually slowed; the respirations become slow and may be of a Cheyne-Stokes type.

In some cases (a comparatively small number) there is evidence of a general septic infection. The temperature remains high throughout the disease or may be very irregular with irregular chills and sweats, but even in these cases the pulse remains slow until late in the disease. In other cases the temperature remains normal or subnormal until the last day or two, when it may ascend to 103° or 104° F. Sudden death sometimes occurs from a rupture of the abscess cavity into the surrounding brain substance. Localization of the abscess is difficult on account of its infiltrating character. When the temporosphenoidal lobe of the brain is affected it may be localized in some cases by a careful examination for aphasia. This, however, implies a fairly good mental reaction of the patient, which is, however, very apt not to be present in abscess complicating middle-ear disease. An abscess in the temporosphenoidal area usually destroys the connection between the auditory and visual speech area; as a result of this when the patient is shown an object of common use, he is not able to recall the name of it, although he may be able perfectly to appreciate its purposes and uses or even in some cases to describe it. If the abscess is sufficiently extensive to destroy the visual areas in the occipital lobe, or the optic radiations transmitting impulses to them, hemianopsia may be present. The extension of the abscess in an anterior direction may involve the motor fibres and produce paralysis of the opposite side of the body.

An abscess below the tentorium in the cerebellum gives rise to persistent vomiting, optic neuritis, marked vertigo, nystagmus, and a very ataxic gait like that of a drunken man. The knee-jerk on the same side

on both sides may be absent, and there may be a marked clumsiness and slowness of movement of the extremities on the same side as the lesion. Facial paralysis may be present, due to the *pressure* on the facial nerve or from involvement of the pons. This may also be due to the local process in the middle ear.

**Diagnosis.**—Where a source of infection can be determined, such as at following traumatism or evidence of a local inflammatory disease in the brain tissues in direct connection with the seat of infection, it makes the diagnosis comparatively easy.

Abscess of the brain complicating middle-ear disease must be differentiated from sinus thrombosis and meningitis. The percentage of cases of abscess, of thrombosis, and of meningitis complicating ear disease varies in each of these from 30 to 35 per cent. Thus in Poulsen's cases of complications of ear disease there were 13 cases of abscess, of thrombosis, and 11 of meningitis.

Sinus thrombosis gives a higher temperature, as a rule, with a rapid rise, with tenderness in the region of the mastoid or of the neck, thrombosis of the deep veins of the neck, cyanosis, and sweating. All the considerations given above under Sinus Thrombosis (p. 938) must be taken into account.

In meningitis the onset is usually much more rapid and the course of the disease much shorter than in brain abscess. The temperature here again is higher, often fluctuating, associated in the early stages with swelling of the pulse, and later with a rapid, irregular pulse, hyperesthesia of sight and sound, persistent headache, twitching of the extremities, rigidity of the neck, retraction of the head, and a purulent fluid on lumbar puncture, or evidence of the infecting agent in the cerebrospinal fluid—i. e., when the connection between the area of meningitis and the spinal meninges remains open.

In traumatic cases brain abscess must at times be differentiated from brain tumor. A beginning or latent brain tumor in a child may show evidence of very rapid growth after traumatism to the head. In a case of a boy of four years recently reported by me, with Dr. J. H. Mason, the following symptoms were presented:

The symptoms were of seven weeks' duration, and began with drowsiness quickly followed by left-sided hemiplegia, headache, restlessness, and night-cries; vomiting and partial incontinence of urine were also present. There was a partial return of power in the left leg after two weeks, and consciousness and speech were preserved until shortly before death. Fever and convulsions were absent. The pulse was 55 on day of admission to the hospital. A few hours later he became unconscious; the pulse became rapid and weak, the temperature rose, and the symptoms of fatal paralytic compression of the brain were present.

The patient was trephined the same night, in the hope that a hemorrhage or a collection of pus might be evacuated, as there was a history of a fall preceding his illness, but nothing was discovered, and he died shortly afterward.

On autopsy, a gelatinous tumor, with areas of hemorrhagic extravasation, was found to occupy the anterior two-thirds of the right hemisphere. It began immediately beneath the ependyma of the third ventricle, and extended to the external capsule. The anterior third of the internal capsule was infiltrated by the tumor-mass. At no point did the tumor reach within an inch of the cortical surface. Microscopic examination showed a neuroglioma, composed entirely of neuroglial nuclei and fibres. The tumor was of central origin, of an infiltrating character, and inoperable.

A careful history of the case will often show evidence of cerebral irritation before the traumatism. The intracranial pressure of a tumor is more marked; the optic neuritis develops early, is much more intense and is present in a much larger number of cases (80 to 90 per cent.). There is no fever and symptoms of local brain irritation are more positive and prolonged than in abscess.

**Prognosis.**—Where distinct abscess formation is present in the brain tissue the prognosis depends upon the cause of the abscess, the extent of brain involvement, and the period of evacuation. In infiltrating abscess complicating middle-ear disease the prognosis is unfavorable, because it is rare in such cases to find the abscesses encapsulated. Oppenheim's statistics show 96 out of 196 cases cured. In traumatic abscess 38 cases out of 60 recovered.

**Treatment.** The treatment of brain abscess is entirely surgical. Evacuation and drainage of the abscess cavity are absolutely necessary. The necessity of early operation should be insisted upon. This is especially true in cases of middle-ear disease. The difficulty of making a positive diagnosis of meningitis or sinus thrombosis should not deter from early operative procedure. When in the course of middle-ear or mastoid disease there is distinct evidence of intracranial involvement, and especially when this evidence points to local brain irritation and is progressive in spite of local treatment, an operation should be done to relieve the local bone condition and to exclude the possibility of sinus thrombosis, meningitis, or abscess. This treatment is necessary in any one of the three conditions named. Exploration of the brain under aseptic procedure should be carefully and thoroughly carried out. Inasmuch as a grooved director may easily slip over the wall of an abscess cavity, an instrument with a sharp point or even free incision with the knife should be employed. In the temporosphenoidal lobe of the brain and in the cerebellum this will do no harm, other than a possible hemorrhage due to the cutting of a vessel which can be easily controlled. Exploration of the cerebellum should never be neglected when an exploration of the temporal sphenoidal lobe gives negative results.

#### TUMORS OF THE BRAIN.

Tumors of the brain are of not infrequent occurrence in childhood. Starr has collected 300 cases under nineteen years of age, as follows:



NATURE OF TUMOR.		POSITION.	
Tuberculous tumors . . . . .	152	Cerebellum . . . . .	96
Glioma . . . . .	87	Pons varolii . . . . .	38
Sarcoma . . . . .	34	Centrum ovale . . . . .	35
Gliosarcoma . . . . .	5	Basal ganglia and lateral ventricles . . . . .	27
Cystic . . . . .	30	Cerebral cortex . . . . .	21
Gummata . . . . .	2	Corpora quadrigemina and crura cerebri . . . . .	21
Other varieties . . . . .	80	Base . . . . .	8
		Fourth ventricle . . . . .	5
		Medulla . . . . .	6
		Multiple tumors . . . . .	43

It necessarily follows from the above classification, and this agrees with my own experience, that tuberculous tumors are those usually met with in children. Gliomata and sarcomata are occasionally met with, the other forms being very rare.

**Pathology.**—Tuberculous tumors may occur as solitary or multiple growths. They are more frequently met with as multiple tumors than any of the other varieties in the above classification. They may affect any part of the central nervous system. They are most frequently met with at the base of the brain between the crura, in the neighborhood of the fissure of Sylvius and near the median line of the cortex. They vary in size from 8 mm. to 4 cm., and show a marked tendency to become encapsulated. The capsule is formed, when the tumor is situated deep in the brain substance, by a zone of proliferated neuroglia cells, and when they occur on the surface, by the thickened, infiltrated, adherent pia mater. The encapsulation is in reality only a pseudoencapsulation, inasmuch as the thickened zone of glia cells is a part of the inflammatory process itself, and as a result of this, attempts at removal usually result in failure or are only accomplished at the expense of considerable uninvolved brain substance. Section of tuberculous tumors of large size may show a very friable interior, but distinct softening or complete breaking down is exceptional. In tumors persisting for a long time partial or complete calcification may occur. In a tumor recently removed at autopsy in a woman aged twenty-six years, at the Henry Phipps Institute, the tumor had lasted from early childhood. Active symptoms of brain tumor were then present, but had subsided leaving the girl completely blind, and with symptoms of cerebellar irritation; complete calcification of the tumor had occurred with small cystic cavities containing a clear fluid within the calcified mass. The involvement of the brain tissue may be by infiltration and destruction of the area involved, but is more frequently a local infiltration with an accumulation of new cells about the central area, the formation of the tumor respecting the nervous tissue and producing disturbance of function by pressure upon it. The microscopic examination gives the raked-field, granular appearance at the centre of the tumor, with a zone of epithelioid and giant cells at the periphery. In active tumors the tubercle bacilli can be demonstrated in the tissues and offer a differential diagnosis between tuberculoma, gummata, and degenerating sarcomata.

Gliomata are infiltrating tumors of rapid growth and develop immediately beneath the gray matter either of the ventricle or of the cortex

and involve, as a rule, large areas of brain substance. The tumor mass is soft, almost pulpy, very vascular, at times infiltrated with hemorrhagic areas and surrounded by edematous brain tissue. There is no attempt at encapsulation, and it is often only with difficulty that the boundary of the tumor mass can be determined by the naked eye at autopsy. Sarcomata resemble in their gross characteristics the gliomata, but are, as a rule, of much firmer consistence and not infrequently encapsulated. This is especially true of tumors of this class growing from the bones of the skull or the membranes of the brain and extending into the brain substance. Sarcomata may also grow from the bloodvessels within the brain substance. Sarcomata may occur as multiple tumors, but not with the same frequency as the tuberculous type of tumors. Very vascular sarcomata sometimes develop within the ventricles of the brain from the choroid plexus.

Cystic tumors of the brain do not differ in their characteristics from these growths met elsewhere in the body. Among these tumors have been described parasitic cysts such as the echinococcus and cysticercus cellulose, dermoid cysts, etc.

**Symptomatology.**—We may divide the clinical manifestations of tumors within the cranial cavity into those caused by an increase of the intracranial pressure and the associated hydrocephalus which is not infrequently associated in children and those due to a local disturbance of function of the particular part of the brain involved.

*General Symptoms.*—We must depend for our diagnosis of the presence of a brain tumor upon certain general symptoms. Not infrequently, and this is especially true in children, the general symptoms of marked intracranial pressure may be the only symptoms presented. This will be the case when the so-called silent areas of the brain are involved or where important functional areas are involved only by pressure and this pressure is of very gradual evolution.

The symptoms presented are of insidious onset and of slow development. Exacerbations may occur and a slowly developing tumor may be transformed into a rapidly growing one by traumatism, or, more rarely, by the development of some intercurrent infection.

*Headache* is an early symptom and persists throughout the course of the disease. It is usually diffuse, although it may be localized to the occiput in tumors of the posterior fossæ of the skull. It varies in intensity; at times of a dull, gnawing character; at others, of an acute agonizing kind, described by the patient as if the brain were being pulled out by sharp hooks. Headaches may be paroxysmal in type and associated with vomiting. In tumors involving the meninges or causing distinct local pressure on the meninges the pain may be distinctly localized and associated with marked tenderness. This, however, should never be depended upon for localization unless the other focal symptoms about to be described correspond. The headache increases in intensity up to the point of maximal intracranial pressure and is usually less severe in the later stages of the disease. Headache may be associated very early in the disease with—

**Convulsions.**—These do not differ from convulsive seizures due to other causes in children. They vary in frequency and in intensity. As a rule, they occur at long intervals, but several may be present in a single day. Convulsive seizures may last only a few seconds or may be prolonged for several minutes. They may be so slight as to cause only a momentary loss of consciousness with slight rigidity, or they may be so intense as to produce marked exhaustion. The general convulsions could be differentiated from the local Jacksonian convulsion limited to one part of the body and due to local irritation of the motor cortex. (*vide infra*.)

**Optic Neuritis.**—The examination of the eyes early in the disease will show either a distinct optic neuritis or a marked congestion and swelling of the disk, which later develops into optic neuritis as the pressure increases. This is present in 89 per cent. of cerebellar tumors, the most common at of tumor formation in childhood. It is present in 80 per cent. of all cases; it may be associated with gradual impairment of the vision, going on to complete blindness. Not infrequently in children sudden blindness occurs. This is, however, probably due to the fact that previous defect of vision was not noticed. All suspicious cases should be carefully examined by the ophthalmoscope even where defect of vision is not complained of.

In a brain tumor in a boy of eight years, recently under my observation, a marked choked disk was present in spite of the fact that the boy spent several hours a day reading.

**Vomiting.**—Vomiting is of frequent occurrence and is seen much more often in children than in adults. It occurs independently of the ingestion of food and later may be unassociated with nausea. The vomiting in some cases is more or less continuous and leads to a very rapid loss of strength. It is sometimes brought on by simple change of position or even movement of the head. In other cases it is dependent on the presence of vertigo. This is often an early symptom and may be slight or very intense. It usually occurs in paroxysms at intervals of longer or shorter duration. During the attack there may be only a slight dizziness where everything about appears to be moving or the patient may have a sensation of turning or sinking and may suddenly fall to the floor during the attack.

As the disease advances distinct mental disturbance becomes manifest. This symptom is present in tumors affecting any part of the brain, but is more marked and develops earlier in tumors affecting the frontal lobe. It is also more distinct where headaches are frequent and intense. There is usually progressive deterioration of all the mental faculties. There is loss of intensity of concentration early associated with the failure of the memory. The child ceases to care to play and manifests during the day a peevish attitude; affectionate children exhibit not infrequently complete indifference to those dear to them. In later stages of the disease distinct torpor develops, from which the child with difficulty may be aroused and very late complete unconsciousness may supervene.

The pulse which in the early stages remains normal or is distinctly slowed becomes rapid late in the disease, the respirations slow and superficial. In older children a humorous or witty cast of reply may be given in response to all questions asked when the tumor affects the frontal lobe. In the early stages of tumors affecting the frontal lobes in young infants before the cranial cavity is completely closed the increase in intracranial tension may lead to hydrocephalus. The skull is enlarged in all its diameters. There is a protrusion of the fontanels and separation of the bones of the skull.

*Symptoms of Localization.*—The focal symptoms of brain tumor, like the general symptoms above described, are of gradual development. The higher development of the left side of the brain of right-handed people and the localization on the left side of the brain of the cerebral speech mechanism makes the localization of left-sided tumors easier and more accurate than those of right-sided lesions. It is often possible to diagnose tumors in the frontal lobes. The development of marked defect of mentality early in the disease and a humorous or pseudo-witty disposition may lead to a presumptive diagnosis of frontal tumor. Loss of memory, irritability, lack of concentration, usually show, in tumors elsewhere in the brain, only when the disease is well developed or even very far advanced, and their early appearance is more frequently found in frontal tumors. This may be confirmed later in growing tumors by involvement of the motor areas. When the posterior third of the third frontal convolution on the left side is affected this will give, in right-handed children, disturbance of speech—motor aphasia. Disturbance of speech develops gradually; at first only a hesitancy due to the loss of use of certain words is observed, followed later by complete loss of expression. Inability to write may be associated when the lesion is sufficiently large to involve the neighboring areas toward the convexity. In some cases the aphasia will only be present when the child is in an erect position and the tumor pressing by its own weight causes disturbances of function in Broca's area. Tumors involving the motor area, either by extension or by primary involvement of the cortex, give rise to Jacksonian epilepsy. Thus, a tumor beginning high up in the motor area and producing irritation of the leg centre gives rise at first to a local convulsive movement usually clonic in character, affecting the leg of the opposite side of the body. This will be associated with loss of power, at first slight, but increasing as the tumor grows. As the irritation of the cortex increases the other motor centres may be affected and the convulsion, at first beginning in the leg, spreads to the arm and later to the face of the same side and may become general, involving both sides of the body. As a rule, when the convulsion is localized to one extremity consciousness is preserved, although it may be lost in very localized convulsions. When the convulsion involves more than one-half of the body consciousness is lost. When the tumor begins in the arm or face areas the convulsion is at first localized to these areas and when it becomes general begins in the area of primary involvement. It is, therefore, of considerable diagnostic importance that the con-



Lesions of brain tumor should be carefully watched and the mode of onset carefully noted. The reflexes are increased and when the motor area is involved the Babinski reflex (extension of the toes) is present, with ankle clonus.

Tumors in the superior parietal lobe are associated with astereognosis or an inability to recognize or name objects by handling them. Tumors of the superior parietal area usually involve the motor area and later produce Jacksonian epilepsy with loss of power of the opposite side of the body. Ataxia and some anesthesia in the opposite arm and leg may be present in superior parietal lesions.

Lesions of the inferior parietal lobe of the left side (supramarginal and angular gyri) produce word blindness. When the letters of the alphabet are shown to the child it is unable to recognize them. Individual letters may be recognized with an inability to understand simple words. The child is able to understand what is said to it and to express itself in ordinary language. If the lesion extends deep in the brain substance it will involve the visual fibres going to the cortex of the occipital lobe and produce loss of vision in the same half of the visual field in both eyes (homonymous hemianopsia). Lesions of the occipital lobe likewise produce hemianopsia and the patient is able to recognize only objects on the opposite side to the lesion. If a drinking cup is brought in front of the child from the blind side of the visual field no attempt is made to grasp it until it passes the median line. If it is brought from the opposite side the child immediately grasps it as soon as it is brought into the visual field. Tumors affecting the temporosphenoidal lobes of the left side produce word deafness—*i. e.*, inability to understand spoken commands. English words to the child like some foreign tongue. There is also loss of memory, or rather the inability to recall the names of people or places, which is due to the fact that the memory of spoken words is stored in the first and second temporal convolutions of the left side in right-handed people.

Tumors lying deep in the brain substance usually produce pressure on the fibres of transmission and result in monoplegia or hemiplegia without localized convulsions, anesthesia on the opposite side of the body, or hemianopsia. Tumors at the base of the brain can be localized by the involvement of the cranial nerves. Each case will have to be studied with reference to the exit points and the intracranial course of the cranial nerves. Tumors of the crus between the pons and the cerebellar hemisphere (the crus) produce paralysis of the third nerve on the affected side by direct involvement of this nerve going to the eye on the same side and paralysis of the face, arm, and leg of the opposite side which have not yet crossed to the opposite side to supply those structures. Tumors of the pons may produce a paralysis of the external rectus with divergence of the eye of the same side due to involvement of the sixth nerve, and paralysis of the muscles of mastication and of sensation on the same side due to involvement of the fifth nerve, or of paralysis of the muscles of the face due to involve-

ment of the seventh nerve, any or all of which is associated with paralysis of the arm or leg of the opposite side of the body. The reflexes, such as the knee-jerk, are often lost in tumors of the pons and of the cerebellum.

In tumors of the medulla, paralysis of the tongue and of the palate, with some difficulty of deglutition due to involvement of the esophagus on the affected side, may be associated with paralysis of the arm and leg of the opposite side, or all four extremities may be paralyzed.

*Tumors of the Cerebellum.*—This is the most frequent seat of cerebral tumors in childhood and the symptoms presented depend upon the part of the cerebellum affected. When the middle lobe of the cerebellum is affected or when the tumor affecting the lateral lobes is sufficiently large to press upon it, or the connection of the cerebellum with the cerebrum through the superior peduncle is interfered with, there results a distinct and marked disturbance of gait. It is first manifested by staggering with a tendency to walk in one particular direction, either to the right or to the left. The patient usually staggers in the direction opposite to the seat of the tumor; he may, however, tend to go toward the tumor; so that this is of little value in determining the side of the lesion. In well-developed cases the gait becomes so ataxic that it resembles that of a drunken person. When the tumor is situated in the lateral lobe of the cerebellum and produces irritation of the cerebellar cortex nystagmus is present. When the tumor develops on the inferior surface of the cerebellum there is an early involvement of the cranial nerves, associated with paralysis of the face (seventh nerve), deafness (eighth nerve), unilateral paralysis of the tongue (twelfth nerve).

**Diagnosis.**—The diagnosis of the presence of a tumor within the cranial cavity will depend upon the course of the disease and a careful consideration of the presence of the general symptoms of increased intracranial tension of slow and gradual development with the presence of one or more groups of localizing symptoms. The conditions from which brain tumor must be diagnosed are abscess of the brain, subacute or chronic hydrocephalus, tuberculous meningitis, and chlorosis.

Brain abscess runs a much more rapid course, as a rule, or after running a comparatively rapid course for a short time the symptoms subside to reappear later from rupture of the capsule following traumatism or spontaneously. The presence of an infecting agent, as suppurative middle-ear disease, or the occurrence of traumatism is an important factor in the diagnosis. The course of the symptoms in brain abscess is relatively rapid, that of tumor slow and gradual. Even where a latent glioma is excited into activity by traumatism the subsequent course of the disease is gradual and progressive, with a predominance of the irritative symptoms, whereas in abscess the evidence of destruction of tissue occurs early. Optic neuritis is of much more frequent occurrence in tumor than in abscess. A slow pulse and subnormal temperature early in the disease are in favor of a diagnosis of abscess. The diagnosis of abscess should not always be made merely because there is a purulent condition of the middle ear or of the mastoid. The course

of the disease and the other factors above referred to should be taken into consideration. Two cases have come under my observation where abscess was diagnosed on account of associated middle-ear disease and tumors were found at autopsy.

Tuberculous meningitis may be mistaken for brain tumor. This is especially true in those cases of tuberculous meningitis running a long course with little fever and with a gradual development of hydrocephalus. The headache, however, is more severe in meningitis; there is retraction of the head, irritation to light and sound, and tubercle bacilli in the cerebrospinal fluid.

Tumor of the middle lobe of the cerebellum pressing on the aqueduct of Sylvius may cause hydrocephalus, and in a case of this type coming late under observation acute hydrocephalus due to inflammation of the lining membrane of the ventricle was diagnosed. In simple chronic hydrocephalus the disease runs a very prolonged course and there may be no localizing symptoms. The extremities may be rigid and ataxia may be present in the arms, but this is always much more marked in tumor at the same stage. If the cranial nerves are involved at all in hydrocephalus it is only late in the disease and is then due to tension rather than to irritation. A careful history of the course of the disease or a careful study of the patient if under observation will make the diagnosis. In the case above referred to in a tumor of the middle lobe of the cerebellum causing hydrocephalus, a re-examination of the history revealed early evidence of local disease in the cerebellum, which was later followed by the hydrocephalus.

Chlorosis may cause severe headaches, defect of mentality, vomiting, vertigo and, in a few cases, optic neuritis, but the absence of localizing symptoms and the evidence of marked anemia, both in the appearance of the patient and on blood examination, should make a presumptive diagnosis, which is later confirmed by the disappearance of the symptoms with the improvement of the blood condition under proper therapy.

The diagnosis of the character of the tumor must be made by taking into consideration the evidence of primary disease elsewhere in the body and the course of the disease itself.

The presumption in tumors of the cerebellum in childhood is that we are dealing with tuberculosis on account of its frequency. The presence of tuberculosis elsewhere in the body is of considerable value. Tuberculous tumors, as a rule, progress more slowly than either sarcoma or glioma. Gliomata are of much more frequent occurrence than sarcomata and are, as a rule, if not always, single growths. Both sarcomata and tuberculous tumors are not infrequently multiple. Where there is evidence of syphilis elsewhere in the body the presumption is that we are dealing with a gumma.

Spontaneous recovery from tuberculous tumors is occasionally seen. In glioma and sarcoma complete recovery never takes place, although a spontaneous arrest or temporary recovery under treatment has been reported. Starr reports two cases of this type, both sarcomata. In one of these the symptoms subsided for a period of four months and in the

other for a period of eight years. The duration of the symptoms of brain tumor varies from several months to several years.

**Prognosis and Treatment.** Prognosis is altogether unfavorable. Less than 10 per cent. are so situated or are of such a nature as to make operation advisable. Of the other 90 per cent., gummata alone yield with any degree of frequency to internal treatment. Not all, however, of syphilitic tumors yield to treatment, and, on the other hand, tumors other than syphilitic not infrequently show marked improvement under antisyphilitic treatment. It therefore follows that in inoperable tumors a course of mercury, preferably by inunction, associated with increasing doses of iodide of potash, should be tried. Syphilitic tumors which yield to medication show rapid improvement after a few weeks. The treatment, however, should be kept up some time after the disappearance of the main symptoms. The improvement obtained in tumors other than gummata by mercury and the iodides is usually temporary.

FIG. 196



Sarcoma of the head.

In inoperable cases headache, vertigo, vomiting, and convulsions will demand attention. For the headache phenacetin or acetanilid may at first be tried, but it is usually necessary to resort to the use of opium. The vomiting, the vertigo, and the convulsions may be relieved by the use of the bromides and a careful regulation of the diet. Where the headaches are very persistent, and there is marked optic neuritis with progressive failure of vision, trephining has been done with good results in some cases. In a recent trephined case under my observation the headaches have entirely disappeared up to the present time, six weeks after the operation, and the swelling of the optic disk has entirely subsided with full vision intact.

**Surgical Treatment.**—It may be stated as a general rule that in tumors of the cerebrum if distinctly localized, progressive in character, and not yielding to medical treatment, trephining should be recommended both



an exploratory and therapeutic measure. It can never be positively stated whether a tumor is or is not removable before opening the skull. In tumors situated beneath the cortex, free incision into the cortex should be made if the position of the tumor cannot be determined by inspection or palpation of the exposed brain. In gliomata and sarcomata attempts at removal usually result in failure. The surgeon should recognize the impossibility of complete removal of infiltrating tumors, and should remain content with the opening of the skull cavity for relief of pressure when such tumors are discovered. It should be remembered that tumors, whether beginning in the bones of the skull or secondarily infiltrating them, are associated with profuse bleeding both from the scalp and from the bones when operation is attempted. For this reason it was deemed advisable to operate on the patient shown in Fig. 196. Secondary infection from the nose was also feared if operation were attempted in this case.

Tumors at the base of the brain in the neighborhood of the fourth ventricle or of the pons, the medulla, or of the optic thalamus cannot be removed on account of the operative difficulties and the functions of these structures themselves.

When operative procedures are necessary valuable time should not be lost in trying to get results from drug treatment. The recuperative power of the patient is better in the earlier stages of the disease, and when the tumor is removable the insult to the cerebral tissues will be less intense and extensive in the earlier stages of growth.

#### INTRACRANIAL HEMORRHAGE.

Intracranial Hemorrhage in children may affect any of the intracranial structures. We have, therefore, to deal with subdural hemorrhages, subarachnoidal hemorrhages, and hemorrhages into the brain substance. In addition to these there are met with in infancy hemorrhages beneath the scalp, and epidural hemorrhages between the bones of the skull and the dura mater.

**Etiology.**—The causative factors in the production of intracranial hemorrhage may be divided into those preceding birth, those operative at the time of birth, and those subsequent to birth. Traumatism to the mother in the late stage of pregnancy has been shown to produce hemorrhage into the brain substance (Gibb). I have in my collection the brain of a fetus of six months, the result of a miscarriage, with an organized, subarachnoidal hemorrhage covering one-half of a hemisphere, which must have existed for a considerable time before the miscarriage. This miscarriage was spontaneous and occurred while the patient was in the hospital. In another case the child was born at term, and at autopsy a grumous, bloody fluid occupied the posterior third of the subdural space above the tentorium on the right side. The hemisphere was intact and covered by the pia and arachnoid, but had developed only two-thirds the size of its fellow-hemisphere. From the character of the

exudate and the failure of the development of the brain this hemorrhage must have taken place comparatively early in pregnancy.

Hemorrhage into the cranial cavity at birth is most frequently due to traumatism, although it may occur in perfectly normal births. The application of forceps with or without fracture of the bones of the skull is a frequent cause. The application of force applied to the trunk or to the extremities in breech presentations or after version may produce the same result. The traumatism may be a spontaneous traumatism due to long and difficult labor. A displacement of the parietal bones with compression of the superior longitudinal sinus in some cases produces a passive congestion, with distention of the veins of the convexity and a rupture of these veins either into the meningeal cavity or into the brain substance.

Compression of the cord or interference with the venous circulation returning from the brain by malposition of the cord around the neck may be a causative factor. It will, therefore, be seen that the hemorrhage at birth in most cases is venous in type, and when arterial hemorrhage occurs there is direct traumatism to the head with fracture or a free disposition to rupture from arterial disease due to hereditary syphilis or other causes.

Hemorrhage after birth and during childhood is most frequently associated with sinus thrombosis above described, or as the result of an inflammatory process of the brain in association with some acute infection. Passive congestion due to tuberculous disease of the mediastinum or hypertrophy of the thymus gland may lead to meningeal hemorrhage.

Cerebral hemorrhage in late childhood, apart from the meningeal hemorrhage due to traumatism or rupture of the middle meningeal or of its branches, is of rare occurrence. Two cases have come under my observation, one immediately following scarlet fever, the other after diphtheria. The probable explanation in both cases was a local inflammatory process involving the wall of the vessels, producing a weakness and subsequent rupture. In both cases the hemorrhage was into the brain substance and in the distribution of the lenticulostriate artery.

#### SUBDURAL HEMORRHAGE.

When the hemorrhage occurs at birth the child is either born dead or in a condition of asphyxia. In rare cases it may be very pale. If artificial respiration be performed and vitality returns definite symptoms are manifested and the child may die in the course of a week or recover with evidence of marked disturbance of cerebral function. The cyanosis usually persists, the temperature is subnormal, somnolence is present, in fatal cases gradually passing into coma. Convulsions are present, but are rarely generalized, and are frequently limited to the eyes and to the face; sometimes an arm or even a whole side is involved. If the child lives sufficiently long, contractures develop. Persistent vomiting and

retention of the intestinal contents are observed in the early cases. Paralysis is rarely met with in early infancy due to hemorrhage. The child usually dies before the end of a week, but the symptoms may persist for several weeks, and where the hemorrhage is slight recovery may take place.

When the destruction of brain tissue or the pressure on the brain interferes with its development, atrophy or sclerosis of the brain may result, leading to one of the cerebral atrophies of childhood. (See Cerebral Palsies of Childhood.)

**Diagnosis.**—The diagnosis depends upon a knowledge of the etiological factors at play during birth, and the presence in the newborn after a difficult labor of marked cyanosis, subnormal temperature, and convulsions. The only other condition from which subdural hemorrhage may be diagnosed is tetanus neonatorum. The absence of cyanosis and of difficult labor and the presence of the tetanus bacillus in the umbilical cord would make a diagnosis.

**Treatment.**—Artificial respiration should be used to overcome the immediate effects of the hemorrhage, and heat and mild stimulation employed to assist in controlling the shock. Surgical procedures in competent hands has given fair results in a small number of cases. The removal of the clot should not be attempted until the strength of the child is such as to stand the shock of a serious operation, but the after-results of hemorrhage are so serious that this method of treatment should be tried more frequently. Attention should here be called to the results of the use of forceps in the production of cerebral traumatism. In cases of difficult or prolonged labor, hemorrhages into the cerebral meninges may occur in the childbearing process, and this may account to a limited extent for the cerebral disorders which occur in a large percentage of cases of forceps delivery. It should, however, be borne in mind that traumatism to the infant brain from careless use of instruments is likely to give rise to serious after-effects. A normal or even somewhat prolonged childbearing process should not be interfered with merely to reduce the pain or discomfort to the mother. While the application of instruments to assist in delivery of the head may be a good routine procedure in normal cases in the hands of expert and careful obstetricians, I feel quite confident that the natural process of childbearing is altogether the safest for the integrity of the cerebral tissues. Children with defective or retarded mental development (conditions which do not attract the attention of parents until late childhood and to which the attention of the obstetrician, as a rule, is never called), give so frequently in our clinics a history of instrumental delivery, that great care should be used in clinics in which the forceps is used as a routine measure to study the effects of this procedure on the cerebral tissues in later childhood instead of being satisfied with immediate results after delivery. Where the use of forceps is clearly indicated, they should be used promptly, and with careful attention to the prevention of too much pressure on the head. Delay in their use may be as serious for the integrity of the cerebral tissues as careless application

in cases where they are not indicated, and where the life of the child and the integrity of the cerebral tissues is not taken into as much consideration as the comfort of the mother.

### CEREBRAL HEMORRHAGE.

Hemorrhage into the brain substance is closely associated with the infectious fevers. The position of the hemorrhage corresponds to that seen in the adult. The lenticular nucleus is usually the seat of the hemorrhage, with pressure on or destruction of the internal capsule and sometimes involvement of the optic thalamus. Hemorrhage into the cerebellum may occur. Hemorrhage into the ventricles is of frequent occurrence in early infancy, and may occur during birth. It is sometimes the result of an infiltrating subarachnoidal hemorrhage into the ventricles through the transverse fissure, or it may be due to the rupture of one of the vessels of the choroid plexus or of a vein immediately beneath the ependymal lining of the ventricle.

**Symptomatology.**—The symptoms produced by hemorrhage into the brain substance varies greatly in infancy and childhood; in late childhood they do not differ essentially from those seen in the adult. There is a sudden apoplexy; the child falls to the floor unconscious, with relaxation of the body and complete loss of tonicity on the paralyzed side. The pupils are usually dilated and may be equal or unequal, but during the unconscious period do not react to light. In hemorrhages into the ventricles the pupils are contracted; there are persistent and prolonged coma and convulsions. The temperature is normal and may be subnormal on the affected side. The unconsciousness may last only a few hours or may be prolonged in ventricular or extensive hemorrhage for days or until death takes place. As consciousness returns the left side of the body is found to be paralyzed, including the lower portion of the face, the arm, and the leg. The reflexes which had disappeared during the unconscious period are now present and increased. After a varying period, depending on the extent of disturbance of the motor fibres, sufficient power returns to enable the child to walk by dragging the paralyzed leg. The return of power develops first in the leg and later in the arm and face. The muscles employed in the finer and more complex movements remain paralyzed. When hemorrhage occurs in the cerebellum the patient presents a cerebellar ataxic gait (see p. 950), with nystagmus, clumsiness of motion on the affected side, with or without loss of power.

Ventricular hemorrhage is usually fatal. The unconsciousness is prolonged and convulsions persistent.

In infancy and early childhood the hemorrhage may be latent with an increasing coma; or be associated (in the majority of cases) with convulsions, contractures, and partial or complete paralysis. It is usually fatal, and if recovery takes place, an atrophic paralysis on the opposite side of the body, with aphasia in left-sided lesions is present.



**Diagnosis.**—In infancy hemorrhage of the brain cannot often be differentiated from other forms of intracranial hemorrhage. In childhood a sudden onset with unconsciousness, complete flaccidity of one side of the body, and a persistent hemiplegia following the more severe infectious fevers will make the diagnosis of cerebral hemorrhage. Cerebral embolism may give the same group of symptoms, but consciousness may not be lost, and there is usually a source for the embolus in disease of the left heart.

**Treatment.**—When the hemorrhage occurs the child should be placed on its back or on the non-paralyzed side, with the head somewhat elevated. If the pulse is full and the face is flushed or cyanosed venesection may be done, with the withdrawal of sufficient blood to produce a depressing effect on the pulse. The relief to the high tension of the circulation, if secured early, will prevent further bleeding into the brain. Free purgation is indicated, and this may be secured by a drop of croton oil on the tongue. The administration of 0.324 gm. (5 gr.) of bromide of sodium and of the same dose of chloral hydrate by rectum should be given for the convulsions. Bromides should also be given if the child is restless. Care should be used in the administration of cardiac stimulants on account of the possibility of producing further hemorrhage.

#### CEREBRAL ATROPHIC PALSIES.

Quite a large number of cases come under the observation of those dealing with diseases of children, of spastic paralysis of cerebral origin. In all such cases a pathological process in very early life leads to extensive disease of one or both of the cerebral hemispheres. While the symptomatology varies considerably, the clinical picture is distinct and easily recognized. Three separate groups depending upon the extent of motor involvement are seen:

1. *Cerebral Spastic Hemiplegia.*—This is due to involvement of at least the motor area of one cerebral hemisphere.

2. *Cerebral spastic quadriplegia, or diplegia*, as it is frequently called, in which all four extremities are involved and due to bilateral involvement of the brain.

3. *Cerebral spastic paraplegia*, in which the legs alone are involved, due to involvement of the vertex of the brain, affecting the leg centres which lie close together on either side of the superior median fissure of the brain.

In any of the above groups the lesion may involve areas of the brain other than the motor areas, with the production of sensory, special sense, or psychic manifestations.

**Etiology.**—The cerebral atrophies of childhood, as their name indicates, are essentially manifestations of disturbance of cerebral function occurring very early in life. The intensity of the psychic and other manifestations, with involvement of only a small portion of the brain mass, is due to the occurrence of the cerebral insult at a period when the

cortical cells have not yet assumed their normal function. Cortical cells may assume under abnormal circumstances, such as the presence of free blood, degenerating brain substance, etc., a metabolic function, and in this way lose that function for which they were primarily intended. I have seen the cortical cells giving a pure hemoglobin reaction where the presence of free blood necessitated its removal from the tissues. I have also seen a condition of the nerve cells years after the primary disturbance of function in such a condition of degeneration as could only be explained by some such original perversion of function. A certain number of cases may be explained by prenatal pathological processes, such as in the two cases of my own mentioned under Meningeal Hemorrhage, in the cases reported by Osler in the brain of the fetus, the mother of which died of typhoid fever about the sixth month of pregnancy, and in a case of Cotard, following an injury to the mother. The effect of syphilis and alcoholism in the parents interfering with the proper development of the central nervous system has been suggested. It is, however, much more likely that a diseased condition of the vessels due to these causes is a more potent factor. Osler reports a case following ligature of the carotid.

The majority of cases undoubtedly result from some disturbance of cerebral function at birth or shortly after birth. A prolonged or difficult labor, and especially where this is associated with the use of instruments or requiring version, is an important etiological factor. Such a history was obtained in 177 of the 400 cases reported by Starr. Where only one child in the family is affected it is usually the eldest, and the history given is that the labor was much more prolonged and difficult than that of the subsequent children. The history of a blue baby, with or without convulsions, and the evidence of the malformed head of large numbers of these children, suggests the occurrence of meningeal or cerebral hemorrhage at the time of birth.

Traumatism to the mother and that due to the childbearing process itself are not infrequent causes of cerebral hemorrhage. I have seen extensive cerebral hemorrhage practically destroying the entire hemisphere as the result of a fall on the head. Twenty-two of Starr's cases gave a history of severe falls during infancy.

In cases developing some time after birth the disturbance of cerebral function may have had its origin at birth or as the result of some pathological process during early childhood. Injury to the soft brain tissues during the childbearing process may not be so great as to cause extensive hemorrhage, and yet may be sufficiently severe by the production of small capillary hemorrhages or by pressure, as to result in the production of symptoms when the child has reached an age when the function of the cortical areas are required or when they would first attract the attention of parents. There is no doubt that extensive inflammatory and toxic processes affecting the brain may be the result of the acute infections, such as pneumonia, scarlet fever, measles, diphtheria, or typhoid. In whooping-cough passive venous congestion during a spasm of coughing may lead to the rupture of a vessel in the brain. Rhein has

recently reported a diffuse encephalitis due to whooping-cough. This condition may also obtain as a result of convulsions, but it is always difficult if not impossible to determine whether the hemorrhage was the result of the convulsion or *vice versa*.

Heredity plays an important role. The heredity is not a direct heredity, but the presence of epilepsy, insanity, grave hysteria, or neurasthenia in the parents.

**Pathology.**—Lesions found at autopsy in the majority of cases can be traced directly or indirectly to some disturbance of the circulation—*e.*, cerebral or meningeal hemorrhage, cyst formation, thrombosis, or embolism. In the other cases a sclerosis with atrophy as a result of an inflammation of the brain or of the meninges or both; hydrocephalus with adhesions at the base, or as a result of inflammation of the lining of the ventricles, and atrophy of the brain from pressure by cystic conditions of the meninges or depressed bone have been found. In all cases independent of the primary cause an atrophy of a part or of the entire brain with primary or secondary sclerosis results. Any part of the brain may be affected. In lesions purely of vascular origin, as thrombosis, embolism, etc., the motor area in the region of Rolando and the cortex in its immediate neighborhood is most frequently involved. Microcephaly (a condition in which a cyst or cysts occupy the cerebral hemisphere) is found in the largest number of cases, as was observed in 132 of the 343 collected by Starr. Whether this condition was primarily of vascular origin or due to simple failure of development could not be determined. The frequency of the other lesions in Starr's cases are as follows: sclerotic atrophy as a terminal result of encephalitis, 7 cases; maldevelopment (failure of development of the cortical cells), 1 case; vascular atrophy, 23 cases; meningoencephalitis, 21 cases; abscesses, 14 cases; intracranial hemorrhage, 18 cases; hydrocephalus, 5 cases.

Not infrequently circumscribed lesions are associated with a failure of full development of the rest of the brain, and a condition of microcephaly results. In the hydrocephalic cases the skull may be larger than normal; not infrequently the dome of the skull on the normal side of full normal contour, while that covering the atrophic hemisphere is smaller and more sloping.

**Symptomatology.** *Cerebral Spastic Hemiplegia.*—When the cerebral injury occurs at birth or before birth the symptoms are present from birth. Where the cerebral disease is not extensive the symptoms may not be noticed for several months. When the cerebral traumatism is marked at birth, the presence of convulsions, cyanosis, and unconsciousness directs the attention to the loss of power and spasticity early in infancy.

In cases developing some time after birth, due to thrombosis, embolism, as a result of a slowly developing sclerosis secondary to traumatism at birth, which, however, may not be manifested by distinct symptoms at that time, the onset is associated with general convulsions and unconsciousness. There may be only one or two convulsions or a series of



convulsions, with prolonged unconsciousness lasting over an entire day or even several days. The convulsions are often described by the mother as inward spasms—i. e., where the tonic rigidity of the body in the unconscious period is associated with slight or periodic clonic convulsions.

The convulsions in some cases recur at varying intervals, the paralysis developing either immediately after the first seizure or after one of the subsequent attacks. It may be progressive, increasing after each attack. The paralysis at first is a flaccid paralysis, but it may be spastic from the beginning. The lower face, arm, and leg are at first completely paralyzed, but as the child grows sufficient power returns to enable it to walk, and in a few cases return of power in the leg may be almost complete. In the vast majority of cases the loss of power remains very marked, and is associated with decided rigidity and some contracture at the knee. This gives rise to a distinct spastic gait, with dragging of the toe and a rotatory movement of the body to swing the palsied leg forward. There is less return of power in the arm, which may be completely useless either from the paralysis or more frequently from the secondary rigidity which keeps the arm flexed at the elbow, the hand flexed on the arm, and the fingers tightly closed in the palm. But even in the cases where there is a fair return of power, and where the contractures are not very marked, certain other associated motor phenomena may seriously interfere with the use of this member. There is often a lack of volitional control; when an attempt is made to extend the arm an involuntary flexor movement results. When an attempt is made to use the arm of the sound side, an involuntary associated movement of a like character develops in the paralyzed arm and interferes with bimanual operations. In a large number of cases (about one-fourth of Osler's series) a rhythmic tremor, which may be very fine or very coarse; a gross, inco-ordinate choreiform movement of an intention type or a slow, constant, snake-like movement of the fingers and arm, due to alternate contractions of different groups of muscles (athetosis), which seriously interfere with the use of the arm and often cause extreme annoyance and discomfort may be present. The paralysis of the lower face is frequently associated with contracture, and this, together with the failure of development of the skull on the affected side, produce a marked asymmetry. Athetoid movements about the mouth may be present.

The paralyzed side fails to keep pace with the growth of the opposite side, and in later childhood the extremities are much shorter, smaller, and with marked loss of vasomotor tone. This is manifested by coldness, mottling, or considerable cyanosis. The reflexes on the paralyzed side are markedly increased and are associated with ankle clonus and the Babinski reflex. While there is failure of development of the affected side there is, as a rule, no true degenerative atrophy. The electrical examination gives normal reactions. The sensation on the affected side is normal, but in rare cases may be lost when the cerebral lesion is very extensive.



*Aphasia* is present in the majority of cases when the lesion is on the left side of the brain and there is a right hemiplegia. It is usually motor in type, the child merely being unable to talk, although it may be able to understand what is said to it and may learn to read and write.

As the child grows it often recovers the faculty of speech, probably through the right brain taking up this function.

*Epilepsy* is of very frequent occurrence and develops in the majority of cases shortly after the onset of paralysis, but may be delayed for months or even years. It should be borne in mind that the weakened nervous system of such children, and even as late as adult life, is very prone to reflex disturbances through peripheral irritation, or manifests disturbance very easily from intoxication. The epileptic manifestations vary greatly. They are of frequent occurrence and more intense than the essential epilepsies. There may be simply *petit mal*, partial epilepsy beginning in an extremity and either localized there or extending to the rest of the body, and associated with or without loss of consciousness, or they may be general convulsions presenting the clinical picture of essential epilepsy. Temporary paralysis in the affected extremities may follow the local or general convulsion. Either as a result of the frequent epileptic attacks or more frequently as a result of the brain lesion there is presented a decided mental weakness.

*Mental Defect.*—This is one of the most marked and distressing symptoms of this disease. All grades of mental defect from a slight lowering of the intelligence to complete idiocy are seen. Idiocy or complete lack of intelligence, with inability to acquire ideas, may be present from the beginning. This is most common in those cases dating from birth or very shortly after birth. In other cases imbecility or the inability to acquire other than the simplest ideas is frequently met with.

In a large number of cases surviving early childhood there is a condition merely of weak-mindedness or retarded development. Some cases show apparently normal mental power, but are unable to stand the stress of advanced education, and when this is persisted in, develop neurasthenia, hysteria, persistent headaches, or insanity. Such children are often irascible, of violent temper, and frequently manifest a tendency to purposeless cruelty to animals or other children, and of destruction of inanimate objects.

Lesions near the motor area, but involving it only by irritation, may produce very little loss of power, but slight or irritative motor symptoms. Thus an increased tonicidity of the muscles on the affected side produces spasms or a tendency to athetoid movements only when volitional movements are attempted. In other cases an athetosis may be very marked, with comparatively little loss of power.

When the temporosphenoidal lobe is affected on the left side in association with involvement of the motor area, deaf-mutism may result.

In a boy of twelve with marked spastic hemiplegia there was deaf-mutism. He could not understand what was said to him, but could hear and understand the significance of the signal bell at the school. This

boy, in spite of frequent epileptic attacks, was of fair intelligence but of very violent temper.

When the occipital lobe is involved hemianopsia may be present in association with hemiplegia. Total blindness is sometimes present. This may be due to lack of development of the optic nerve or defective development of the cortex in the occipital area.

*Cerebral Spastic Quadriplegia.* In this group all four extremities are affected. It is invariably a condition dating from birth, and the clinical picture presented is the same as that above described under Hemiplegia, with a bilateral involvement instead of unilateral involvement. It results from extensive injury to the brain, affecting both hemispheres. In severe cases there is rigidity and contractures of the extremities, with rigidity of the back and neck muscles early in infancy, and if the child lives it remains a bedridden idiot. In less severe cases the child learns to walk, but the contractures and the athetoid movements give a peculiar, shuffling, clownish element to the gait, which usually excites ridicule. In the mild cases there is spasticity of the gait and a certain rigidity and clumsiness of arm movement, which in some cases improves as the child grows older, and in other cases grows steadily worse. In the severe cases idiocy or imbecility is the invariable rule. Distinct and definite mental deficiency is present in all cases. Convulsions, violent attacks of temper, and even maniacal outbreaks, aphasia, and irritative motor disturbances are more frequently present in this group than in the hemiplegias. All grades of motor speech disturbances, from slight stammering to complete motor aphasia, are seen. Not infrequently, in training children who stammer and stutter, cases are met with in which a clumsy, awkward method of elevating the arms to carry out the breathing exercises attracts the attention, and on careful examination they often give a history of traumatism at birth, some rigidity of the muscles leading to the clumsiness and increase of the reflexes. As a rule they are backward children, who have not sufficient mental power to keep pace with normal children of their own age in the public schools.

A diseased condition very closely resembling this, but due to defective development of the motor tracts, with symptoms of atrophy, has been described on page 914; but in these children the manifestations are purely motor, with rigidity, contractures, loss of power, and increased reflexes, but with no evidence of cerebral disturbance. These children are as bright intellectually as other children. There is no history of traumatism or of difficult labor or of other evidence of cerebral insults at any time in the history of the child. Several children of the same family are sometimes affected.

*Cerebral Spastic Paraplegia.*—In this group the lower extremities alone are affected. The lesion is confined to the apex of the brain, affecting both leg centres or the fibres originating therein. The traumatism is localized and in most cases of vascular origin due to hemorrhage, or in connection with lesion of the superior longitudinal sinus or branches of the anterior cerebral artery (the arm and face centres on either side

are supplied by branches from the middle cerebral). The same history of difficult or prolonged labor, followed early in childhood by a spastic weakness or paralysis of the lower extremities, without loss of sensation, disturbance of the bladder or rectum, and with some mental deficiency, and, in some cases, with epilepsy, is presented. This should be differentiated from spinal lesions where there is disturbance of sensation up to the point of lesion, involvement of the bladder and rectum, disease of the spine itself, with normal intelligence, and without epileptic attacks. (See p. 897.)

**Diagnosis.**—Any one of the above groups is easily recognized by the history of the onset in early childhood, the distribution in a hemiplegic, quadriplegic, or paraplegic form of paralysis, spastic type, with increased reflexes, lack of sensory disturbance, mental deficiency, and epileptic seizures.

From acute anterior poliomyelitis, whether affecting both lower extremities or an arm and leg of the same side, the diagnosis can be easily made by the history of an acute onset, with a flaccid type of paralysis, loss of the reflexes, and reactions of degeneration in the paralyzed muscles, and, later, by the atrophy in the muscles affected. In these cases there is no aphasia, no mental deficiency, and no epilepsy.

High spinal lesions producing paralysis of all four extremities, due to fracture or dislocation at birth, Pott's disease after birth, myelitis, tumor of the cord, or hemorrhage into the cord, may be differentiated from the cerebral spastic quadriplegia by the evidence of disease of the bone, either by direct examination or by means of the x-ray; by the sensory disturbance below the point of lesion, the involvement of the bladder and rectum, and the absence of cerebral symptoms.

**Prognosis.**—The prognosis in the vast majority of cases is unfavorable. Apart from the motor disturbances—paralysis, epilepsy, etc.—which incapacitate for manual work, the mental deficiency is such as to either delegate the sufferer to an insane asylum or institution for feeble-minded children. If less marked it incapacitates him for serious mental work, and renders him dependent on others for care and support. A very few cases are able to pass through a common-school education, and even in rare instances successfully take up a college training and assume the duties of professional work, but the experiment is always a dangerous one on account of the tendency to develop under stress some serious psychosis—epilepsy, insanity, neurasthenia, etc. The motor paralysis is not only persistent in all but a few cases, but often develops with advancing age. Where the cerebral traumatism is slight improvement may occur. The epilepsy of this disease rarely yields to treatment.

**Treatment.**—In the severe cases treatment is of little avail. Sooner or later it is necessary to send the patients to a home for the feeble-minded or to an asylum. Where a moderate amount of intelligence is present, they are best treated in special training schools for the feeble-minded under the care of expert and skilled teachers. Where the intelligence approaches normal, the education of the child should be very carefully guarded; better under individual teachers than in large classes where

the stress to keep pace with normal minds may work serious consequences. The lack of moral tone in many of these children, often amounting to criminal propensities, also interferes, with too close association with other children. Parents too often, I might say almost invariably, lavish an affection, care, and expenditure on these deficient entirely disproportionate to the results which may reasonably be expected, and often to the exclusion and harm of their normal and deserving children, to be repaid, even in those cases who stand education well by ingratitude and often disgrace. A quiet, simple life in a country home, with proper nutrition and well-directed discipline, gives by far the best results.

Contractures may be prevented and the use of the paralyzed limbs improved by intelligent massage and passive movements. Where contractures have occurred, section of tendons not only relieves the deformity but often cures painful spasm in the affected muscle. Orthopedic apparatus is useful in some cases in maintaining a good position and to prevent increasing deformities. Its usefulness in this disease, however, is very limited.

The history of surgical procedures directed to relieving or curing the cerebral condition is not such as to warrant trephining in many cases. I have never seen any appreciable results. When acute cerebral hemorrhage can be diagnosed, and especially when this is of a meningeal type, operation at the time, on account of the hopeless nature of the sequela, would be justified. The only case in my personal experience in which this was done the result was unsatisfactory on account of the death of the child, and no conclusion could be drawn from it. In later childhood, operation is only justified when there is distinct evidence of local pressure on the brain tissue. These cases form a very small part of the number coming under observation, and most of them are found to be due to cystic conditions of the meninges and of the cortex. Even in such favorable cases it is very exceptional to have sufficient relief of symptoms as to justify an operation. The best result that can be expected, where the mental condition is markedly deficient, is to raise the grade of the imbecility. It is questionable, however, inasmuch as we cannot effect a cure, whether it is not better to permit these unfortunates to remain as near intellectual oblivion as possible, instead of elevating them to an appreciation of their own deficiency and suffering.

#### HYDROCEPHALUS.

In dealing with this subject I shall only consider internal hydrocephalus: an accumulation of fluid in the ventricles of the brain causing pressure on the brain substance when the skull is already closed, and producing extension of the skull, separation of the bones, and enlargement of the head when it occurs in infancy. Internal hydrocephalus may be either a general internal hydrocephalus, with distention of all the ventricles, or a partial hydrocephalus in which the fourth ventricle



not involved. Hydrocephalus may be congenital or acquired. Of the acquired form we have to deal with the chronic internal hydrocephalus and an acute internal hydrocephalus—the meningitis serosa of Quincke. **Acute Internal Hydrocephalus.** **Etiology.**—While an acute internal hydrocephalus is a frequent accompaniment of tuberculous and other forms of meningitis, it is occasionally met with as a pathological condition due to an inflammation localized to the lining membrane of the ventricles and of the choroid plexus. Quincke, however, considers the acute effusion into the ventricles as comparable to the serous effusion into the skin in angioneurotic edema. In a series of experiments which

FIG. 197



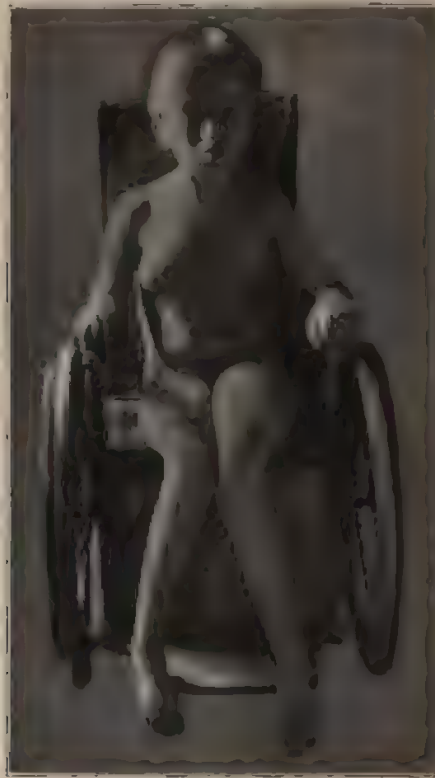
Hydrocephalus.

carried out a few years ago into the nature and pathology of this affection, the results confirmed in certain respects this idea; extensive inflammatory lesions of the ependyma were produced without hydrocephalus. The inflammatory lesions seen in the ependyma are the result of a toxic condition of the retained fluid. The disease is more frequently seen in late childhood and adult life, and frequently in those who show some previous hydrocephalus. Traumatism may also be a factor.

**Symptomatology.**—The symptoms develop acutely with slight fever, which gradually ascends for several days and then slowly drops to normal,

and after a short intermission is again followed by another paroxysm which may be repeated several times throughout the course of the disease. At the onset there is evidence of marked increase of intracranial pressure. Headache develops early, and is associated with choked disk and blindness, retraction of the neck, slowing of the pulse, somnolence, stupor, delirium, and coma. Paralysis of the cranial nerves may be present. All of these symptoms, which are most intense with the fastigium of the fever, subside, and may entirely disappear when the

FIG. 196



Hydrocephalus with paralysis of both extremities, wrist-drop, etc.

temperature drops to normal, to reappear again in the course of several days or a week with reappearance of the fever. This is repeated for several paroxysms, the patient either becoming progressively weaker and dying with symptoms of intense intracranial pressure, or the successive paroxysms decrease in intensity, and the patient goes on to convalescence with partial or total blindness and weakened mental power. The symptoms have entirely disappeared after lumbar puncture, with the evacuation of a large quantity of fluid. Cases have been reported running an afebrile course with the same variations in symp-

toms and, in some cases, without variation, and which could not be differentiated in their acute form from brain tumor. Many of the rapid recoveries from brain tumor, so diagnosed, may belong to this category.

**Diagnosis.**—The diagnosis from meningitis and brain tumor may be made by the recurring paroxysms of fever, with the variations of the clinical picture during the febrile and afebrile period. The absence of a causative agent in the cerebrospinal fluid and the relief of symptoms after lumbar puncture also point to hydrocephalus.

**Treatment.**—The treatment is that of meningitis, with the use of lumbar puncture as a therapeutic agent. In the case reported by Dr. Burr and myself from the Philadelphia Hospital the variations in the symptoms, and their almost complete absence with the subsequent decline of fever, led us to postpone any radical method of treatment. In cases where lumbar puncture gives negative results, tapping of the ventricles should give good results. Prognosis in the majority of cases is unfavorable. Evacuation of the ventricular fluid in all but a very few cases gives the only hope for relief and cure.

**Chronic Internal Hydrocephalus.**—In the congenital form the head is enlarged at birth, and not infrequently gives rise to difficult labor, and may necessitate surgical procedure to deliver the child. The cause of hydrocephalus in the fetus is not definitely known. The changes in the choroid plexus and the ependyma are not sufficient to account for the distention of the ventricles. Czerny explains this condition by pathological changes found in the adrenal, and which he thinks causes a disturbance of the cerebral circulation. The frequency with which spina bifida, defective development of the bones of the spine, polydactylia, webbing of the fingers, etc., are found in children with hydrocephalus, would lead us to consider it a structural developmental defect rather than the result of a local process.

In the acquired form inflammatory lesions obstructing the connection between the ventricles of the brain and the subarachnoid spaces or lesions closing the aqueduct of Sylvius are found in a small percentage of cases. In a large class of cases there is no obstructive lesion and nothing is found to account for the hydrocephalus. The thickening of the ependymal lining of the ventricles and a sclerotic condition of the choroid plexus have led some to consider the hydrocephalus the result of a previous inflammatory disease of these structures. There is, however, little to support such a contention.

The accumulation of fluid in the ventricles may be enormous, reaching several pints. The brain tissue in mild cases may be fairly well preserved; in severe cases it may be represented by a thin band of tissue one-fourth to a half-inch in thickness. The bones of the skull are very thin, with separated sutures; or when union has taken place Wormian bones are found in them.

**Symptomatology.**—In the minor grades of hydrocephalus the only symptom present may be a certain grade of mental deficiency. While normal intelligence and even precocity have been found in hydrocephalic

children, they are certainly very rare; and while I have seen one case of such intelligence as to enable the boy to take a college degree, his mental condition was certainly not one to be envied. The child learns to walk late, if at all; in a great many cases a spastic type of paralysis is present from the beginning or develops after the child has learned to walk. Epilepsy is present in a large number of cases. The course of the disease is more or less progressive in early life, but may be spontaneously arrested or even decidedly improved after operative procedures.

**Diagnosis.**—The diagnosis is merely a matter of observation. The large, globular head with protruding forehead, small, receding face, deficient mentality, and one or more of the complications referred to, makes the diagnosis easy. The diagnosis from the rachitic type of head need only be mentioned.

**Treatment.**—There is no medicine that has any appreciable effect in causing a disappearance of the fluid. Mechanical measures, such as the use of compression by adhesive bands, etc., are no longer employed. Operative procedures, such as tapping the ventricle and draining into the subdural space (Taylor) sometimes produce good results, and is being tried by a number of careful observers, but it is rare that a completely normal mental condition is obtained.



## SECTION XII.

### DISEASES OF THE SKIN.

By CHARLES TOWNSHEND DADE, M.D.

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#### CHAPTER XXXVIII.

ECZEMA—ERYTHEMA—URTICARIA—IMPETIGO—SCABIES.

It is not my intention to study all of the diseases of the skin that may be seen in infancy and childhood, but to present merely the clinical characters and treatment of the manifestations that most often fall under the notice of the physician who is called to treat children. For this purpose no classification is necessary, but as Eczema is the most common of the lesions of the skin it will be first described and then the other diseases in the order of their relative importance.

#### ECZEMA.

Eczema is a simple exudative inflammation of the skin characterized by erythema, vesicles, papules or pustules, attended with itching, the production of more or less infiltration and thickening of the skin consequent upon the serofibrinous exudate that takes place from the dilated bloodvessels, with a final stage of scaling or crusting. As in all inflammations of this type, the tissues return to their normal condition on subsidence of the disease. Eczema may be acute, subacute, or chronic; one form not necessarily running into another, but starting as one or the other as such at the outset. More often in children we meet with the acute or subacute types, but by the continual exacerbations and relapses, in point of time, the disease, in such cases, may be said to be chronic. Eczema in infancy does not differ essentially from eczema in adult subjects. Certain types of the disease, however, may be more constantly seen in infants and young children and have more definite sites of election; also the disease in them may be more rebellious to treatment, more irritable and more liable to recurrence, but the pathological process is the same in all and the term *infantile eczema* has no particular significance. Children under five years of age are the more frequently attacked, and the larger proportion of cases up to this age occur during

the first year of life, the head and face being with them by far the commonest sites of the disease (Fig. 199).

A greater tendency for eczema to be pustular is noted in infancy; also its more ready production, in those in whom a predisposition exists, by local irritations as well as reflexly through disturbances of the alimentary canal. Eczema in older children differs in no respect in its essential manifestations from that occurring in adults. Children of all ages, but particularly those of early years, present a greater tendency to enlargement of the lymph nodes, but the frequency and intensity of the adenopathy occurring in eczema will depend largely upon the care with which ports of entry are protected from the invasion of pyogenic micro-organisms.

FIG. 199



Eczema. (Photograph by Dr. Dana Hubbard.)

**Etiology.**—The etiology of eczema is not precise. No child has ever been born with an eczema, and yet while eczema cannot be said to be an inherited disease, as such, the predisposition to it does exist and exists, too, as a prime factor, in infantile eczemas as well as in the production of all true eczemas. It is hardly going too far to say that without a tendency to the disease, inherited or acquired, no true eczema can be produced. The *artificial* and *traumatic* eczemas, so-called, are but simple inflammations—dermatites, which on removal of the exciting causes and with, or even without, simple, appropriate treatment readily subside. These same causes, however, acting on a predisposed skin may produce a very different condition, which continues long after the exciting element of its causation has been suppressed, is more or less rebellious to treatment, and a true eczema is recognized produced in a

subject with an underlying eczematous tendency without which it is safe to say this disease could not have been brought into existence—a dermatitis, yes, but a true eczema never.

Children with a tuberculous predisposition; those in poor surroundings breathing bad air; ill-fed, anemic children with poor assimilation are in a condition especially favoring germ invasion, and when exhibiting affections of the mouth, nose or throat, with cervical and submaxillary adenopathy, present the type known as lymphatic, strumous, or scrofulous. They have a particular predisposition to eczema, yet it cannot be said that there exists a tuberculous or scrofulous eczema, in the true sense of the word, any more than exists a *gouty* eczema, which is merely an exhibition of eczema in an *also* gouty subject. A tendency to eczema is said to be one of the most frequent manifestations of gout during infancy. Children with rheumatic antecedents are also liable to the disease. The commonest exciting cause, the prime factor almost, in the production of eczema in infants and young children lies in relation with the derangement of the function of digestion, whether due to the food itself—quantity or quality—its administration or its assimilation.

While no one of these factors may be considered absolute in producing eczema, it is more probably brought about by *overfeeding* than by any other cause in connection with the food, and it is in the fat, healthy-looking, overfed infant, whether it be nursed or artificially fed, that we meet most frequently that familiar, violently itching, florid form of eczema of the face that has won for it the term "infantile." Poorly nourished, flabby children seldom have this form of the disease, as it takes in them a more asthenic type—dry, scaly, and scattered in patches and not very irritable; the itching being very much less marked a feature. In marasmic children eczema rarely discloses itself if at all. It is with overfed mothers' children, who by reason of the easy assimilation of the maternal milk receive too large quantities of it before it causes any definite digestive disturbance, and defects of elimination here play the important part. The role of dentition in the production of eczema has been exaggerated. An eczema may appear long before teething begins, and there is no valid reason for forming a definite type under the title "dentition eczema," for the process of dentition may be in some babies as painful and irritating as one could imagine it without producing any skin manifestation if there be not at the same time other underlying conditions, chief of which is the individual predisposition.

Teething may aggravate the eruption by interfering with the general health, but is never a sole cause of eczema. The same may be said of vaccination; it may light up for the first time an attack of eczema in predisposed subjects or excite exacerbations or recurrences of the disease in such eczematous children. To avoid these possible accidents, children in the French hospitals are vaccinated during the first week or so of life, early infancy being regularly immune to eczema. For external causes acting directly upon the susceptible skin we have most commonly heat, cold, dry winds, too much washing, or washing with

hard water and the use of strong, irritating soaps. The disease may be brought about by bad hygiene of the skin and lack of proper care, especially with regard to the diaper. Contact with irritating, altered discharges from ear, nose, and mouth causes it. An eczema of the upper lip is almost invariably due to a nasal discharge which must be corrected before hope of cure. The irritation from scabies, pediculosis, and other parasitic affections of the skin may finally, in favorable subjects, result in the production of eczema consequent upon the scratching induced by the intense itching of these affections, and finally various micro-organisms, if not its source, may be the cause of the continuance of the disease.

*Reflex irritation* is not infrequently an associated cause of eczema.

Dentition, as a cause, may be said to come under this head; and also irritation from intestinal worms, but their association with digestive disturbances must not be forgotten. Genital irritation from smegma confined by a long or tight prepuce may be the exciting cause of an eczema which will yield to no treatment until the foreskin is trained back and the part daily cleansed of the confined, irritating secretion. Where this is not possible by reason of a very long and tight prepuce, circumcision is necessary.

**Varieties.**—Eczema taking the type of any one of its characteristic lesions may be denominated erythematous, vesicular, papular, or pustular—the four primary forms of the disease. It is not to be understood, however, except possibly in the case of erythematous eczema, that any one of these forms of the disease is limited sharply to the particular lesion which titles it; papules may become capped with vesicles which, through secondary infection by pyogenic micro-organisms, become pustules, the lesions breaking down and by extension forming a weeping, reddened area denuded of epithelium over which is soon formed a yellow or yellowish-green crust resulting from the drying of the exuded serous fluid and pus, and blackened sometimes with blood drawn from the raw surface by the scratching which the intense itching induces. Thus the gamut may be run in any one patch, the final stage of all eczemas, the squamous, in turn succeeding before recovery takes place.

Eczema, while rarely general or universal, is apt to be, apart from purely external causes acting locally, more or less symmetrical. This symmetry is fairly constantly seen in facial eczema. (See Plate XXVIII.)

*Erythematous eczema* in its most typical form is met with most frequently occurring on the face. Beginning at any point as bright or dull red, smooth patches, slightly elevated if at all, with ill-defined borders, it may remain in this patchy state with trifling subjective symptoms, or the whole surface of the face may be rapidly involved, accompanied by a feeling of tension with considerable burning or itching or both, and swelling; the eyes at times being completely closed owing to the facility with which edema takes place in the loose cellular tissues of the eyelids. Later there is but slight scaliness and the surface is dry, remaining so throughout unless by scratching or rubbing the skin becomes broken and oozing is induced. When it occurs on apposed surfaces, as around



PLATE XXVIII.



Eczema.



the genitals, there may be moisture through friction and maceration. The course of erythematous eczema is extremely variable, sometimes yielding quickly to the simplest treatment only to shortly appear anew as bad as ever, remaining with varying intensity from day to day until it fairly becomes chronic, thickened and indurated, and yielding only to the most constant and energetic treatment.

*Vesicular eczema*, while one of the most common expressions of the disease, we rarely see in its typical form, for the reason that, owing to the ease with which the vesicles break, by the time it comes under observation of the physician the tiny superficial vesicles closely aggregated on a reddened base have already ruptured spontaneously or been broken by friction or scratching, and only a more or less profusely weeping, erythematous surface is seen. It is not the contents of the ruptured vesicles alone which constitute the discharge, but a subsequent continuous oozing from the denuded area of a clear plasmic fluid which stiffens the linen and stains it yellow. The discharge drying rapidly on exposure to the air, forms light-yellowish granular crusts. The affected area, which may be quite extensive or confined to small patches, is seldom well defined in contour, the borders fading imperceptibly into the surrounding healthy skin. Infiltration, though slight at times, is always present and can be appreciated by pinching up a part of the affected skin, which will be found thicker and more resistant than normal. The itching is most severe and a child, unless restrained, will often by scratching lacerate the affected part cruelly, rendering it a bleeding mass before relief is thus obtained. This intense itching and the gummy-like exudate staining and stiffening the linen are the two chief characteristics of this form of eczema and can hardly confuse the picture with anything else. This form of eczema occurring on the head and face of infants is known commonly as *milk crust*.

*Papular eczema*, formerly classed in the lichen group of skin diseases under the title of *lichen simplex*, is a common and obstinate form. The papules are from the size of a small to a large pinhead, round or acuminate, bright red in color as they first appear, later dull red or violaceous, and remain as papules throughout as a rule. They may occur in small, fairly well-defined groups which, running together, form large, irregular, infiltrated patches by the coalescing of the individual papules, or the papules may remain discrete and be scattered irregularly over sites of election, the extensor aspect of the arms and thighs and the trunk being the favorite places for the eruption. This form of eczema is, of all the varieties, the most intensely itchy and the summits of the papules are often seen capped with a minute blackened crust, a result of the drying up of the droplet of blood the violent scratching brings to the torn surface. Papular eczema is a dry form of the disease and remains so throughout unless sufficiently irritated by scratching or rubbing to induce oozing and weeping. It is more often seen in the older children and adults.

*Pustular eczema* may supervene upon any one of the other types of eczema as the result of secondary infection by pus cocci or the lesions

may be pustules from the start; itching is not of so aggravated a character as accompanies the other phases of the disease. Pustular eczema generally occurs upon the head and face, being most often seen in poorly nourished children whose surroundings and hygiene are not of the best. When occurring on the scalp the hair becomes matted down by the dried secretion and thick, dirty, closely adherent crusts are formed from beneath which at the edges the confined pus makes its way and, drying, lends itself to increase the crust until at times a considerable area is involved. In cases of some duration there is often loss of hair, which is not permanent, from the long standing inflammation and pus bath to which the part has been subjected.

**Diagnosis.**—The diagnosis of eczema in children presents no especial difficulty, particularly that form seen so frequently on the face and head of infants and young children. These fat, apparently healthy youngsters, with their fat cheeks red and weeping or crusted with dried exudation, the inflammation extending in some cases so as to include the forehead, chin and ears, with the nose and area around the eyes and mouth left free, giving thus the appearance of a mask with the centre cut out, present a picture so typical that it could hardly be confounded with any other condition. Eczema occurring around the buttocks and genital region of infants is generally fairly confined to the area of the diaper; syphilis of this region extends farther along the limbs down to the feet, the soles being often involved; besides the characteristic snuffles and other evidences of syphilis would generally be present to help out the diagnosis, as in any other form of eczema for which syphilis might be mistaken. Papular eczema in its intense itchiness and character of lesions may be taken for scabies, but the situations and distribution of the eruption of the latter would help to clear up the difficulty together with its manifestation in other members of the family. In infants who are nursing from a scabies infected mother, aside from the typical places, the face and scalp, from close contact with the breasts, may present the characteristic burrows and scattered eruption of scabies, and likewise the feet and buttocks from the infected hands of the mother may be similarly involved. In older children these evidences of scabies are more manifest in the situations commonly affected by the itch mite; the flexures of the wrists, skin between the fingers, folds at the margins, anterior surface of the body, the inner surface of the thighs, and the sheath of the penis. In all these places, if carefully looked for, the characteristic burrows may be discovered from which with care an acarus can be picked. While in all these situations papular eczema, too, may occur, its lesions are more often grouped and patchy, not so irregularly scattered as in scabies. Papular eczema and a papular form of urticaria may be mistaken for each other, but the presence of wheals in the latter would clear up the difficulty. In prurigo, the history, its more general distribution and chronicity, together with the characteristic inguinal adenopathy, would differentiate it from papular eczema. Pustular eczema of the scalp closely resembles impetigo of this region, but the latter, when set up by pediculosis capitis, its most common cause,



is almost exclusively confined to the occipital region and nape of the neck; eczema of the scalp is not so limited; besides, nits would be discovered if not the pediculi themselves, and, furthermore, treatment would be quickly decisive. The characteristic isolated lesions of contagious impetigo, looking as if stuck on the sound skin, some of which always occur outside of a larger main patch, would determine the diagnosis between this disease when on the face and pustular eczema. The microscope would clear up any particular difficulty should it occur between the diagnosis of ringworm of the scalp or body and eczema.

**Prognosis.**—Eczema if left to itself untreated runs on, as a rule, indefinitely, showing but little tendency to spontaneous recovery, especially during the early years of life, when at times, even under the most painstaking care, it persists in a disheartening way to those concerned. Ultimate cure, though, can be brought about by judicious local treatment combined with attention to the diet and correction of chronic indigestion and constipation when these exist. Every effort should be made to seek out and eliminate any and all underlying causes. Otherwise any more than temporary relief can hardly be hoped for by local measures and the constant recurrence and lighting up through weeks and months of what seemed at times almost a conquered disease will tax the skill of the physician and patience of the mother to the utmost, to say nothing of the torment by itching to the bearer of the disease. Oddly enough the general condition and spirits of many infants with a persistent eczema of the face seems but little affected; they go on gaining weight and appear in nowise the worse for the violent paroxysms of itching during restless nights, while the mother or nurse is worn out by the watchful attention the little sufferer requires.

**Treatment.**—That an eczema should be treated there ought to be no question and, indeed, the old idea of its being a vent for poisonous material in the blood and its suppression causing other worse (but unknown) diseases to spring into existence is entertained but little to-day and only by those of but the most meagre intelligence upon the subject. Always terminate an eczema as speedily as possible, especially when on the face, if only from a cosmetic point of view; doing so will never prove dangerous and the other organs of the body will go unharmed. If it "strikes in" and will only *stay* in, so much the better for all concerned. In approaching an eczema, whether in children or adults, with any hope of successfully treating it, the aim should be to determine the exciting cause and remove it; then further seek to put the body in such condition that the underlying tendency may be less responsive to the exciting stimuli, external or internal, which produce the eczema. Until this be fairly accomplished hopes of a permanent cure are futile, for local treatment alone will afford but temporary benefit at best on all but a very small majority of cases. There can be but little difficulty in ascertaining what may be the external causes, for by observation and by question of those in charge these may be readily discovered, and while the internal causes are generally due to some disturbance of the digestive tract, or related to a functional disturbance of the liver or kidney,

it is not always at first that one can put his finger on just the exact condition responsible. Investigation of the food in every particular relating to it is of the first importance, for it is in the errors of diet that the most fruitful sources of eczema in children will be found. Examination of the mother's milk at the outset, if the child be nursing, will save time and obviate a speculative groping in the dark as to whether excess of the proteid or the fats be causing the trouble. Regulation of this should be brought about by attention to the mother's diet, seconded by having her take systematic out-door exercise, which alone at times will so alter for the better the quality of the milk that a marked improvement will often be noted in an hitherto obstinate eczema of the face of a nursing infant. In older children, up to three years of age, especially those allowed to come to the table, *overfeeding* is the common error together with injudicious food. In clinical practice, upon questioning mothers as to what the child eats, a common answer is: "Any and everything, just what there is," and, one might add, and at all times. If such children be limited to milk alone for a week or more a marked change for the better will often be noted in an eczema that formerly, under the same treatment locally, had proved most resistant. All children with an eczema should be given plentifully of water between meals, it facilitates assimilation and is better than drugs for constipation. It is useless to lay down precise rules for feeding, what may agree with one will not agree with another, and the diet that suits best can only be found out by experimenting in each individual case. In general starchy food, especially cereals and potatoes, should be eliminated and sometimes even meats during the active stage of an eczema. The regulation of the bowels is of the greatest importance when constipation exists, getting rid of this stumbling block is more than half the battle in many cases. Calomel in doses of 0.0065 gm. ( $\frac{1}{16}$  gr.), three times daily for fat babies, is of the greatest service, and in older children, used in purgative doses, two or three times in ten days, will go far toward relieving the congestion of the face. The bowels should not only be opened but kept open daily, and if necessary by drugs, the milder laxatives, such as the mixture of rhubarb and soda (U. S. P.) alone or in combination, and cascara may be used, always giving plentifully of water throughout the day between meals. Other drugs, such as arsenic and antimony, have but a traditional value as specifics. Whatever may be the temptation to use arsenic, at least let it not be yielded to during the acute stage of an eczema. Cod-liver oil in poorly nourished children is often of use and the syrup of the iodide of iron, wine of iron, and bitter tonics are of value in anemia.

In general management the first importance is the constant protection of the skin from contact with the air; a dressing left off and the skin exposed for several hours will often undo days of treatment. A child with eczema of the face properly protected may be taken out in any sort of weather to which it ordinarily is exposed and be the better for it. In removing crusts, poultices of starch jelly applied when cold and renewed every few hours will be found very efficacious and soothing.

or strips of flannel soaked in sweet oil left on overnight, covered with rubber tissue, will loosen up the crusts so that they may be readily removed the next morning, not by washing with soap and water, however, but gently cleared away with a soft cloth dipped in oil. Water, much less soap, should never be allowed to touch an acute exuding eczema. In removing particles of former applications which adhere to the surface, as when stiff pastes are used, oil answers every purpose, and with care every trace can be removed without undue injury. That an absolute exclusion of water from *all* eczematous surfaces is essential is a mistaken idea; in erythematous patches where the surface is dry and in papular eczema there is no reason for doing away with the benefits of the daily bath. Soap and water energetically applied with hard scrubbing even enters into the treatment of some forms of eczema, and in cases where extensive surfaces of the body and limbs are involved, a rather prolonged immersion in water kept at a comfortable temperature and softened with bran or starch will be found extremely soothing and grateful to the irritated skin, often securing immunity from scratching for hours at a time and if used at bedtime affording a quiet night. Where proper attention can be had there should be no necessity for tying children's hands to prevent scratching; doing this only irritates them the more in their attempts to get their hands free and makes them more restless. The physician should be able to cope with this and it is his duty to supply means of relief and the attendants to employ them at any and all times required, for scratching and tearing the skin must be obviated at any cost. Where constant attention is not feasible the most humane method is to use cardboard splints at the elbows so that the hands, though free, cannot reach the face. Anodynes for the relief of itching should not be even thought of.

Strict attention to cleanliness should be enforced, the diaper should be removed as soon as soiled and replaced by a clean one. Stearate of zinc powder, medicated or not, should be dusted on; it affords the best protection to the skin, it is more adherent than most powders, and, being non-absorbent, the urine is prevented from coming in contact with the parts to any great extent.

Notwithstanding the importance of internal treatment, some form of local treatment, if only as an adjunct to the former, is nearly always necessary, and some forms of eczema indeed are cured by local measures alone. At the outset it will be well to bear in mind two general principles as set forth by Van Harlingen with regard to the local treatment of eczema. These are, first, that in the acute form the treatment can hardly be too soothing; secondly, that in the chronic form the treatment (within limits, of course) can hardly be too stimulating. To avoid to some extent the confused notions as to local applications that result more often than not from just giving a list of prescriptions with general suggestions as to their use, I think it simpler to consider the various forms of eczema, together with location, and taking up a type, as far as possible, give directions that suit it which may be followed out more or less in similar cases.

In the presence of the familiar picture of an acute vesicular eczema on the face of an infant with the inflamed skin oozing and crusted, the first thing to be done is to remove the adherent crusts so that whatever is to be used later may come in contact with the diseased surface. This is best done by applying a cold starch-jelly poultice as before stated, and then by applying pledgets of lint soaked in sweet oil. Another method is to lay on the crusted surface strips of flannel soaked in sweet oil, covering these with rubber tissue, binding them well on and allowing them to remain in place overnight; the following morning the surface can be readily freed from the crusts with sweet oil and made ready for the next step. Soap and water *should not* be used to remove the crusts. So that the inflamed skin be exposed to the air for as short a time as possible after cleansing, a mask made of absorbent gauze should have been previously prepared spread and ready for immediate use with the following paste:

R—Acid. salicylic.	. . . . .	0.10 gm.	(gr. 29).
Amyli.			
Zinc. oxid.	. . . . .	dd 8.00 gm.	(3i).
Ung. petrol.	. . . . .	q. s. ad 80.00 gm.	(3j).—M.

This protective mask is to be well bound on, openings having been made for the nose, eyes, and mouth. Several layers of absorbent gauze must be used in making the mask and the paste spread on evenly to the thickness of 3.2 mm. (fully  $\frac{1}{8}$  inch) thick. This dressing should be kept on day and night and renewed twice in the twenty-four hours. After each removal of the mask and before making a fresh dressing the face is to be freed of adherent particles of the former dressing by the use of sweet oil. In this paste the amount of salicylic acid may be diminished or omitted entirely according to the irritability of the skin; in most cases it can be used freely as above. The practical use of this paste is as follows: The vaselin is largely taken up by the absorbent gauze, leaving a more or less porous mass which absorbs the exudation as it comes from the weeping surface; hence simply smearing the paste thinly on or using it without a covering of gauze defeats the purpose for which Lassar devised it.

The dressings with this paste may be found all that is necessary to a cure; if not, and to complete it more stimulating treatment be required, one proceeds to the use of tar. It is always a delicate question to decide just when tar is to be used, but, as a rule, it should only be employed after exudation has entirely ceased and sound skin has formed. To get the proper benefit from tar compounds they must be rubbed in, not merely layed on. The following is a good compound in which the amount of tar may be varied to suit the case, trying smaller amounts at first and on limited areas to get the effect desired:

R—Oleo cadini	. . . . .	4.0 gm.	(3i).
Ung. zinc. oxid.	. . . . .	q. s. ad 80.0 gm.	(3j).

This is to be gently worked in and appropriate dressings made.



Acute eczema of the above type occurring on any part of the body may be treated on the same lines.

In cases where there is no weeping or but very slight oozing the following lotion may be used at the start:

a {	Zinc. oxid. . . . .	40 parts.
	Pulv. cretæ. . . . .	20 "
b {	Lot. plumbi,	
	Ol. lini. . . . .	ad 20 parts.

The ingredients of *a* and *b* are to be mixed separately and then the two together.

This lotion will be found most efficacious and as it dries quickly and is very adherent it is not readily rubbed off and no outside covering is necessary—a great advantage. It is to be removed with oil.

In pustular eczema of the scalp the head is to be freely anointed with

R—	Acid. salicylic. . . . .	1.65 gm.	(gr. xxv).
	Ol. amygdal. dulcis . . . . .	30.00 gm.	(℥j).

and bound up in flannel cloths covered with gutta-percha tissue or a rubber cap until all crusts and scabs can be removed, continuing the salicylated oil for a few days until the hyperemia and pustulation are abated; then the oil of cade up to 4 c.c. (1 dr.) to 30 c.c. (1 oz.) of sweet oil or vaselin can be applied.

White precipitate ointment from 1.3 gm. (20 gr.) up to 4 c.c. (1 dr.) to 30 gm. (1 ounce) of vaselin will be found useful, but this ointment must be carefully made to get full benefit. Pastes and stiff ointments are to be avoided on the scalp unless the hair be closely clipped.

In papular eczema ointments are generally to be avoided and lotions used. One that has proven the most generally useful is the following:

R—	Acid. carbolic. . . . .	2.92 gm.	(gr. xlv).
	Zinc. oxid. . . . .	5.85 gm.	(℥ss).
	Glycerin. . . . .	9 25 c.c.	(℥ss).
	Aque rose . . . . .	q. s. ad 120.00 c.c.	(℥iv).

The amount of carbolic acid in this may be diminished or increased. Lime-water may be used in place of rose-water. This lotion will be found most efficacious in allaying itching in general; where burning is the more pronounced element the following will be found better:

R—	Calamin. prep.,		
	Zinc. oxid. . . . .	ad 4.0 gm.	(℥j).
	Glycerin. . . . .	8.0 c.c.	(℥ij).
	Aque rose . . . . .	q. s. ad 120.0 c.c.	(℥iv).

This will be useful also for the burning and smarting of beginning erythematous eczema and wherever a soothing application may be needed.

For patches of chronic eczema where there is thickening and induration of the skin the use of tar can be instituted at once, beginning with varying strengths of tar ointments up to the pure oil of cade. More



are confined chiefly to the extremities, particularly the lower. The face and scalp are seldom attacked and the mucous membranes never. The course of the disease is acute, running from ten days to two weeks up to the formation of the crusts, after this the length of the process of repair depends upon the extent of the ulceration that has taken place beneath the crusts. By autoinoculation and the continuance of the cause the appearance of new lesions may persist almost indefinitely. Inoculation with the pus from an ecthymatiform pustule always produces a similar pustule, and I have produced, experimentally on myself, pustules through the fifth generation, the original pus having been taken from a fresh lesion on a child. Each succeeding pustule was smaller than its predecessor, and beyond the fifth one reinoculation proved abortive, the power to reproduce seeming to have died out, or possibly the soil became unsuitable to the growth of the specific germ. Cultures resulted in the demonstration of but the ordinary staphylococci and streptococci, which, however, when inoculated give various results.

The lesion of ecthyma has a very regular and definite evolution. In a few hours after inoculation a small, red, itchy point appears, which increases in size up to three-eighths of an inch in diameter by the second day, when a minute pustule appears in its centre; by the fourth or fifth day the full development of the ecthymatiform lesion is established in the form of a yellow pustule the size of a small split pea, seated on an indurated base circled by a whitish ring of loosened epidermis a sixteenth of an inch in width, marking the advancing area of pustulation, outside of which again is a bright-red areola a quarter of an inch or more in width. Throughout the succeeding days all these elements of the lesion advance—the two encircling bands keeping about the same width, the pustule increasing in area up to the ninth or eleventh day. Drying then begins at the centre of the pustule, which flattens down into a black or brownish crust, still surrounded by the whitish ring of pus-loosened epidermis and the outside red areola. The process may stop at this point and healing take place in from fifteen to twenty days, leaving a superficial cicatrix, with more or less brownish-red pigmentation which slowly disappears. Sometimes the process extends, the advancing area of pustulation being marked by the whitish ring of loosened epidermis, the crust becomes larger, the ulceration more extensive, and a lesion of considerable dimension may be attained. There is a rare and more destructive variety of ecthyma entirely peculiar to very young children and infants—the *ecthyma téoébrant*, *ecthyma ulcéreux* of French authors, and allied to, if not identical with, the gangrenous lesions described as following varicella, measles, etc. (Duhring), under various titles, viz., infantile gangrenous dermatitis, *ecthyma gangrenosa*, *varicella gangrenosa*, etc. It is characterized by the formation of papulopustules or quite large pemphigoid bullæ of brief duration, under which develop circular or oval, sharply defined, punched-out ulcers, surrounded by a slight erythematous areola; the ulceration spreads rapidly, superficially, and in depth, penetrating at times through the derma to the subcutaneous fat; the edges of the ulcer are indurated and considerably

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raised, giving a crater-like form to the ulcer and a depth to it more apparent than real. This is well portrayed in Plate XXIX. The infection spread by autoinoculation, may give rise to numerous closely aggregated lesions, which, coalescing, form large, polycyclic patches. The lesions of this severer form of ecthyma, though found on the buttocks, thighs, anal region, back, and abdomen, are chiefly situated on the upper posterior parts of the thighs and buttocks where the diaper comes in more intimate contact with the skin, for it is chiefly through maintenance of filth in this connection that these parts are so abundantly affected. Accidental lesions through secondary infection may be found on any part of the body, even the scalp, and not infrequently the mucous membrane of the mouth has been involved. This form of ecthyma, while grave, is not necessarily fatal, and the lesions, though often remaining stationary for long periods, heal slowly, leaving indelible scars.

**Diagnosis.**—Ecthyma may chiefly be distinguished from impetigo, with which it is most often confounded, by its more distinctly pustular nature—ecthyma is always pustular. It further differs by the greater depth of its lesions, the inflammatory areola, and whitish line of undermined epidermis surrounding the pustule or crust. Impetigo nearly always occurs on the face, with characteristic stuck-on, yellow, honey-like crusts, ecthyma on the extremities with flat, blackish-brown crusts surrounded by an extensive inflammatory areola.

A furuncle differs from ecthyma in its more extended and vivid redness, greater tumefaction of the tissues, its central core, and greater pain.

From pustular eczema ecthyma may be distinguished by the scarcity of its lesions; their occurring, as a rule, discretely; the size of its pustules; their inflammatory, firm base and external areola.

**Treatment.**—Ecthyma, as a rule, is easily controlled by proper treatment. The first efforts should be directed toward putting the patient in as hygienic surroundings as possible, with daily attention to cleanliness, bathing, and fresh air; the diet should be looked into and made as fully nutritious as possible. Tonics, as in combinations of iron, arsenic, quinine, and strychnine; the syrup of the iodide of iron and cod-liver oil may often be used with benefit, and in some cases are indispensable. Local treatment is of great importance. If parasites be acting as exciting causes, these should first be done away with. After having removed the crusts from the lesions by antiseptic poultices, soaking in sweet oil, or by prolonged alkaline baths or water dressings of carbolic or bichloride of mercury, all source of reinoculation may be removed by thoroughly bathing the excoriated or ulcerated surfaces with 1:60 carbolic acid solution or 1:1000 bichloride of mercury solution or stronger, followed by some constant occlusive dressing, the object of this being as much a cure as a preventive against possible reinfection by scratching. For this purpose a white precipitate ointment spread on cheese-cloth, and exactly fitting the lesion and bound on, may best be used; 4 gm. (1 dr.) to 30 gm. (1 oz.) of the ammoniated mercury in vaselin is none too strong, or the ordinary mercury plaster may be bound on. Pustules should be opened and treated in the same way;



PLATE XXIX.



Ecthyma.



dressings should be made twice daily—the parts being thoroughly cleansed before reapplying the ointment. Various other drugs, such as calomel, aristol, iodoform, naphthol, etc., may be used in ointment or powder form, but the white precipitate will be generally found all sufficient. If healing be slow and the sores sluggish they may be touched with pure carbolic acid, solutions of silver nitrate, or the stronger silver point itself. In the deeper ulcerative forms of ecthyma, where there is a gangrenous tendency, astringent lotions should be used, later coming to the mercurial ointment. One of the best lotions for this purpose is the following:

R <sup>x</sup> —Alum.	5 parts.
Plumbi acetat.	25 "
Aque.	500 "

Ecthyma, as a rule, terminates most favorably, except for the scarring, and it is only in the most neglected cases, following depressing general conditions, where deep and extensive ulceration has taken place, that the disease is at all grave, and even here not necessarily fatal if proper change of conditions be provided and treatment instituted and carried out.

### URTICARIA.

Urticaria is an angioneurotic disturbance, manifesting itself ordinarily by the rapid production in the skin of swellings or "wheals," accompanied by itching, burning, and tingling.

The affection announces itself by an intense itching and the appearance of the characteristic wheals, constituting the familiar "Hives" or "Nettle Rash." The fever and other disturbances which may accompany an acute onset of urticaria have more to do with the underlying cause of the attack than with the eruption itself, ordinarily only the distressing itching and burning mark the variation from the normal condition.

The wheals appearing in successive crops may be very generally distributed over the body or be confined to certain portions—the face, shoulders, neck, arms, thighs, or abdomen—these being the more usual seats for the development of the lesions. They appear, in the common type of urticaria, as fairly prominent elevations of the skin, with sloping, irregular borders, velvety to the touch, varying in size from 19.05 mm. ( $\frac{3}{4}$  inch) or less to 3.175 cm. ( $1\frac{1}{4}$  inches). The color of the wheal is at first pink or red, may remain so, or, later, change to white, depending upon the intensity of the serous infiltration in the derma. Generally discrete, the wheals may become confluent and form extensive patches. The transitory nature of the urticarial lesions is their essential characteristic; they appear and disappear with almost equal rapidity, leaving one place to suddenly spring up in another, effacing themselves without the least trace of their existence, except at times a slight pigmentation. The duration of an individual lesion varies from a minute or two to several hours. The intense itching, which is a pretty constant accompaniment of an urticarial outbreak, is increased on exposure of

the surface to the air, and is generally most marked at bedtime, thus causing in some cases distressing nights of restlessness. Beyond the loss of sleep, which may become serious in prolonged cases and affect the health of the child, the general condition remains undisturbed.

*Papular or Papulovesicular Urticaria.*—This is a variant from the common type of the disease and is peculiar to young children, occurring more frequently during the first few years of life, and, as a rule, in those who are illy cared for and poorly nourished. It is the lichen urticatus, strophulus, varicella prurigo, and infantile urticaria, etc., of various writers. Not infrequently it is mistaken for the rare disease prurigo of Hebra. It is an obstinate form of urticaria and generally worse in summer. The lesion is a papule, induced by inflammatory changes supervening upon or coexisting with the serous exudate in the skin; capped at times, if the inflammation be sufficiently intense, by a vesicle. The eruption occurs in successive crops as millet seed to small pea size, rosy red, acuminate papules, which appear, as a rule, suddenly, and instead of disappearing in a few hours persist several days or longer. They occur more particularly on the upper part of the trunk and the external surface of the arms and legs; though never very large in number, they may be generally dispersed over the body at large or irregularly grouped, and confined to a single locality, such as the external surface of the leg or anterior surface and sides of the thorax. The itching is intense, and owing to the scratching the tops of the papules become excoriated and small, blackish blood crusts are formed which, falling after a few days or so, leave pigmented macules which slowly disappear. Occasionally vesiculation, if sufficiently intense, goes on to the formation of bullae, constituting the *bullous urticaria*, but which, however, should be looked upon more as a complication, and a rare one, than forming a distinct variety of the disease. These bullae, when occurring, are generally limited to the hands and feet of children, and may become pustular through unfavorably hygienic conditions favoring infection.

**Etiology.**—With a predisposition as a groundwork for the production of an urticarial outbreak the secondary causes may be external or internal. Chief among the former are insects and body parasites, and they should always be sought for as a cause in children. The main cause, however, in children is some derangement of the digestive tract, whether temporary and brought about by the ingestion of some improper article of diet, or, through want of efficient treatment, allowed to persist and develop into a chronic intestinal catarrh. Intestinal worms are frequently a cause of urticaria in children. It is a question whether dentition alone plays any part as a cause.

**Diagnosis.**—The diagnosis of the ordinary form of urticaria is simple when in the presence of the characteristic wheals; in their absence the story of the sudden appearance and disappearance of what is said to "look like mosquito bites" will generally give a safe working clue to the trouble. The papular form, being more persistent, may resemble the secondary lesions of scabies very closely, but the finding of the burrows of scabies and its lesions between the fingers and in the other



favorite seats where urticaria is seldom located would serve as a guide in the right direction; except in infants in arms scabies does not occur on the face. As scabies may lead to an urticaria it not infrequently happens that the two are associated, when the difficulty naturally becomes greater and the latter be overlooked unless by inquiry the history of wheals is elicited. When vesiculation takes place in the papule, varicella may be resembled, but the spindle-shaped lesions upon which the easily ruptured vesicle of varicella is seated and other marked features would determine the difference. In case of a severe papular urticaria in very early infancy it might be a question of the rare disease prurigo, and this perhaps could only be determined as time went on or suggested by the severity and persistence of the eruption. Urticaria does not *run into and become* prurigo, but it is often a forerunner of the latter disease.

**Treatment.**—In the instance of an acute attack of urticaria, depending upon the ingestion of some irritating article of food, an emetic may be given if the case be seen early enough; this often will cut short an attack, and nothing further is necessary beyond careful attention to diet. If not seen in time it is best to administer a good dose of castor oil and sweep free the alimentary canal. In the more established forms, where a chronic intestinal catarrh seems to be at fault, a strict attention to the diet is of paramount importance. Starting out with a purely milk diet for a varying period will often modify the eruption of a papular urticaria considerably; then the choice of such articles of food that best agree will be a matter of experiment, more or less, as one goes along; ordinarily sweets should be cut out entirely, and starches, such as oatmeal, greatly limited. Acid fruits, especially strawberries, should be avoided. The bowels should be kept open by small doses of calomel or castor oil, and a plentiful supply of water should be drunk throughout the day; this alone is often the best correction against constipation.

Salicylate of soda and salol will be found useful with the mineral acids, after meals, for the associated indigestion, and the standby rhubarb and soda is most helpful. Antipyrin and quinine in fairly large doses will be found efficacious in children as antipyretics, especially the former, as quinine is difficult to administer to young children without combining it with some syrup that will still further upset the stomach.

For the relief of itching and the general discomfort local measures can hardly be dispensed with, and for this purpose demulcent baths will be found very grateful to the skin: 454 gm. (1 pound.) of starch is sufficient for the ordinary bath; bran can be added to the water for the same purpose. Baths should be warm, not hot, and the body dabbed dry rather than rubbed, and then thoroughly dredged with starch powder or the dolomol powders, which adhere best. Spraying with chloroform is excellent; sponging with aromatic vinegar, diluted extract of witch-hazel, or a saturated solution of bicarbonate of soda will be found useful. The lotions should always be warmed and applied frequently. A good application is a solution of starch boiled to about the consistency of liquid glue, to a pint of which has been added 4 gm. (1 dr.) of zinc oxide, and 8 c.c. (2 dr.) of glycerin. This applied

when cool will often afford the greatest relief. One of the best lotions is the following:

R—Acid. carbol.	. . . . .	8.70 gm.	(gr. 217½).
Zinc. oxid.	. . . . .	6.54 gm.	(3℥).
Glycerin.	. . . . .	10.00 c. c.	(3℥ss).
Aq. rose.	. . . . .	q. s. ad 120.00 c. c.	(3℥v).

Dusting powders at times may be found all sufficient in mild cases; one of the best is the dolomol-camphor, 10 per cent. Heavy, irritating flannels should be avoided and, if possible, soft linen worn next the skin at night. The coverings should be as light as possible. All parasites should be carefully searched for and vigorously eliminated.

Fresh air and tonics and attention to the general condition will in the end go farther toward a cure than most efforts in this direction.

#### IMPETIGO.

Impetigo is an acute inflammatory disease peculiar to childhood, characterized by the rapid formation of very superficial, easily broken vesicles or blebs, the serous or seropurulent contents of which on escaping coagulate and form characteristic granular, yellow, honey-like crusts, without areola, covering an excoriated surface which heals without the production of cicatrices. It is contagious and sometimes epidemic, autoinoculable as well as experimentally so, and is due primarily to the action of a special microbe—the streptococcus of Fehleisen. The frequency with which children are attacked may be accounted for by the delicacy of the skin of the face, the favorite seat of impetigo, rather than by the assumption of any predisposing cause further than that which may be instituted in them through their poor surroundings and general ill nourishment, leaving them with poor defence against attack. In this way eczema and all diseases due to animal parasites may be said to be predisposing causes by provoking the scratching and laceration of the skin which provide a port of entry for the special germ. Though impetigo may occur in any child, it is rare and only accidental in those whose surroundings are cleanly and whose skins are properly cared for. It is a self-limiting disease and individual lesions have an evolution of from ten days to two weeks, but by autoinoculation new lesions may continue to appear and the disease be thus prolonged indefinitely unless means be taken for its extinction.

**Symptomatology.**—Constitutional disturbance is, as a rule, entirely wanting. Contiguous lymph nodes are sometimes swollen and painful. Itching, while not a regular symptom, may be present, and, though slight, is sufficient to cause scratching and thus fresh inoculation is brought about. The primitive lesion of impetigo is an erythematous spot varying from an eighth to a quarter of an inch in diameter, rapidly increasing up to a half or three-quarters of an inch and very slightly if at all raised above the surface of the skin. In a few hours the uppermost, horny layer of the epidermis covering the erythematous spot becomes loosened

PLATE XXX.



Impetigo.





by the effusion of a clear serum, which later may become slightly cloudy, but which at first is always clear, giving rise to a flattened, irregular, partly filled, and hence wrinkled-looking, superficial bleb. The lesion now looks very much like a blister caused by a slight burn of the second degree. These two stages being of short duration, are not always seen by the physician. Very soon, owing to the thinness of the covering membrane, the bleb is ruptured, either spontaneously or by scratching, and a clear, honey-like serum exudes plentifully, which, coagulating, covers the area of the bleb with a granular, heaped-up, amber-like crust with *no* surrounding inflammatory areola (or if present, an extremely slight one), and looking as if "stuck on" the sound skin. It is in this, the more durable stage, that the disease is most often observed; the face, more or less covered with the discrete, characteristic, "stuck on" looking crusts, which may, at times, form quite extensive patches from agglomeration of individual blebs; but outlying lesions in all stages may generally be found in the neighborhood of the larger patches after the disease is once established. The succeeding stage of repair follows on; the crusts become dryer and fall off, exposing shiny red areas exactly corresponding to the deposited crusts. This redness gradually disappears, leaving no cicatrix or subsequent trace of the disease. While the face is the usual site of impetigo the disease occurs behind the ears, on the hands and legs, and sparsely and abortively on the body. It also occurs on the scalp in disseminated plaques, matting down the hair and subsequently causing its fall, which, however, is only temporary. These *postimpetiginous* bald spots are sometimes confusing, being taken for lesions of alopecia areata or evidences of ringworms; they present, however, no element of contagion. (See Plate XXX.)

There is a rare and sporadic form of impetigo occurring in early life which differs only from the ordinary type in that the lesions are larger, better filled, and more distinctly bullous. The bullæ rise abruptly from the healthy skin with only exceptionally a narrow red areola, depending upon the purulence of the contents. They are small in number and occur most frequently over the buttocks, thighs, and pubes, though other parts of the trunk and limbs may be attacked, as well as the face. These bullæ closely resemble ordinary pemphigous lesions, and in all probability the cases reported from time to time of *acute pemphigus* in infants are but examples of this bullous type of impetigo.

**Diagnosis.**—The diagnosis of impetigo presents no particular difficulty in view of the characteristic features of its lesions—*e. g.*, their discrete dissemination over exposed surfaces—face, head, and hands; their having no inflammatory areola around them, and the inoculability of the contents of the blebs and exudate under the crusts. Scabies and varicella may be readily distinguished from impetigo by a comparison of their lesions with the above points.

Pustular eczema of the face may closely resemble impetigo when the lesions of the latter have run together to form patches, but the itching and the larger and inflammatory patches of the former, with green or blackish crusts, will aid in the diagnosis; furthermore, there are nearly

always individual typical outlying lesions in the neighborhood of a patch of impetigo.

Ecthyma may be distinguished by the pronounced inflammatory areola, indurated base, and blackish, flat crusts, covering distinctly ulcerated surfaces. Ecthyma lesions are also painful.

**Treatment.**—The treatment of impetigo is simple and most efficient: removal of the crusts and the use of an antiseptic dressing. The majority of the crusts may be loosened and detached by bathing with hot water and soap; others more firmly adherent may be first soaked in sweet oil overnight. After the removal of the crusts is accomplished the exposed surfaces are to be bathed with a saturated solution of boric acid in water and an ointment of ammoniated mercury varying from 0.65 gm. (10 gr.) to a 4 gm. (1 dr.) to the 30 gm. (1 oz.) of vaselin or rose-water ointment kept constantly applied. At times, in the early stage of the crusts, after their removal there will be noticed a continued exudation of serum from the exposed surfaces. In such cases dab on frequently during the day, with an absorbent cotton tampon, the following lotion:

℞—Camphor-water to saturation and filtered . . . . .	600 gm.	(℥xx).
Sulphate of zinc . . . . .	7 gm.	(2½ gr. xlv).
Sulphate of copper . . . . .	2 gm.	(3m).

It is important that the camphor-water be well filtered. This lotion will sufficiently dry the erosion so that at night the ammoniated mercury ointment may be applied. Encountered in the initial vesicular stage the loose covering of the blebs should be cut away with scissors and the exposed surfaces lightly frictioned with the above-mentioned lotion several times daily—no further treatment being necessary in these early cases.

### SCABIES.

Scabies is a communicable disease of the skin due to the invasion of the upper layers of the epidermis by an animal parasite, viz., the *acarus scabiei*.

It is no longer even the comparatively rare disease in this country, as has been but recently held, for it has become now a fairly common complaint, and this is due rather to an actual increase, as shown by clinical statistics, than to the disease being more frequently correctly diagnosed.

**Symptomatology.**—The manifestations of the disease may be divided into primary and secondary lesions. The primary lesions constitute the pathognomonic characteristic of scabies and consist of the burrow formed by the female acarus as she travels along under the epidermis, feeding and depositing her eggs. At the further end of the burrow may be discerned a small, white elevation, denoting the female acarus beneath the epithelium, and if this be broken carefully and the point of a needle inserted she may be withdrawn clinging to the end of the needle as a tiny, white speck just about visible to the naked eye. The burrows

PLATE XXXI.



Scabies.





appear as fine, white, grayish, or blackish lines slightly elevated above the skin surface; they may be straight or wavy in outline, sometimes S-shaped or in the form of a horseshoe, and vary from an eighth to a half an inch or more in length. The acarus chooses by preference the parts of the body where there is apt to be both warmth and moisture and where the skin is most delicate; hence the burrows are found in such characteristic places as between the fingers and along their sides near the web; the flexure of the wrists, particularly at the inner side; the palms of the hands, feet, and buttocks in infants; inner side of the thighs, anterior border of the axillæ, and in males the genitals. The face is never attacked except in infancy, and then generally through contact with the infected breasts of the mother. These burrows, more or less pronounced, with a white, elevated point at one extremity, constitute the essential and pathognomonic lesions of scabies; and were it not for the intense itching caused by the irritation in the skin as the acarus tunnels its way beneath the epidermis, there would be no others. It is due to the scratching for the relief of this intense itching that the *secondary* lesions supervene. These are produced not only at the sites of election, as noted above, of the burrowing acarus, and naturally in these situations in greater abundance, but, pretty generally, through reflex irritation, over the whole front of the body, barring the face, except, as stated, in infants, and consist for the most part of papules, more or less excoriated, and vesicles, vesicopustules, and pustules. It is this conglomeration of lesions, together with the burrows, that constitutes the eruption known as *scabies*. In cases of some standing the disease may be complicated by ecthymatous and impetiginous lesions, furuncles, etc., and in predisposed subjects by eczema and urticaria. The itching is most pronounced, and is characteristically intensified at night when the patient is warm in bed—the time when the acarus is most actively at work. (See Plate XXXI.)

**Diagnosis.**—The diagnosis of scabies should present no particular difficulty, but it is a strange fact how often the eruption in a long-standing or well-marked case is mistaken for syphilis, to which it would seem, to one who has seen anything of the two diseases, not to bear the faintest resemblance. Not infrequently a patient will be encountered who, presenting a body absolutely free from any eruption, will complain of itching at night, this itching having increased during two or three weeks; a close inspection may or may not reveal the burrows on the hands or elsewhere. In such a case, suspicion having been excited by the history of itching at night, the diagnosis at this stage may be made in two ways: either by treating the hands alone for two or three days, with a resulting discontinuance of the characteristic itching at night; or by letting the disease run on and waiting for the eruption produced by scratching to develop—it surely will in from two to four weeks in full feature. Ordinarily the patient is presented at a stage when the acarus has multiplied and been transferred to other parts from the hands, and the secondary eruption is already present in its polymorphic character cattered over the arms and hands and the front of the body, from a

## DISEASES OF THE SKIN

level of the axillæ to the middle of the thighs. In children the hands may be fairly peppered with pustules, vesicles, and papules, more pronounced at the web of the fingers, where a pustulous eruption is always strongly indicative of scabies. In infants the eruption may appear on the face and head from contact with the infected breasts of the mother; likewise, burrows may be found on the feet and buttocks of infants, having been transferred from the hands of the mother or nurse. The face of children is much more liable to acute inflammation, and in them malar lesions are more commonly and extensively established, either directly due to the irritation of the burrowing parasite or to the impetigo and ecthyma induced by scratching. In a well-marked case the scattering of the lesions, chiefly on the hands, wrists, axillæ, and genitals in males, will distinguish scabies from eczema, for there is more apt to be grouping of the lesions into patches in the latter disease.

**Treatment.**—Scabies is an entirely and readily curable affection; only tempestuous and overtreatments are to be guarded against, for often these two errors set up a resulting eczema or dermatitis more difficult to combat than the original trouble.

Sulphur is the chief and efficient remedy. In the following combination an ointment may be obtained which has stood a long test as regards efficacy and minimum risk of resulting irritation of the skin, both adults and children:

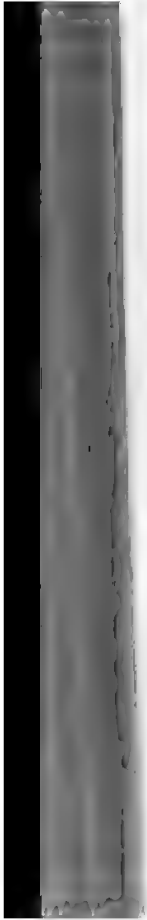
R—Creolæ prep.,						
Sulph. sublim.	. . . . .	46	1.20 gm.	(gr. 2½)		
Bal. Peru.	. . . . .		.61 c. v.	(℥ss)		
Sapo. virid.	. . . . .		.60 gm.	(gr. 8)		
Petrolat.	. . . . .	q. s. ad	90.00 gm.	(℔ij)		

The method of procedure is as follows: having separated an ounce of this ointment into three parts, a warm bath is to be taken at bedtime, lasting from twenty minutes to half an hour, during which the body is well soaped and scrubbed, particular attention being paid to the hands, between the fingers, and folds of the wrists. In mild or beginning cases the hands alone may be treated. After the preliminary bath, in an ordinary case, one part of the ointment is to be thoroughly rubbed all over the body, working it well in, especially between the fingers, and over the wrists, axillæ, and genitals—the rubbing to be done before a fire if possible. Fresh sheets and night clothing having been provided, the anointed patient retires, and the next morning is to omit washing the body, and to put on fresh underclothing. The following night a second rubbing is to be made without a preceding bath, the third night the remaining portion of the ointment is to be utilized, the bath again being omitted. The fourth morning a general cleansing bath is to be taken and fresh underclothing put on, replacing that worn during the course of treatment. Usually these three successive rubbings will complete the cure. At any rate an interval of some days should be made before undertaking another course, should this be suggested by a continuance of the itching, and a soothing lotion used to allay the irritation, due either to the treatment or continuance of the previous inflamed condition of

the skin, consequent upon scratching. The following lotion is best for this purpose:

℞—Acid. carbol.	. . . . .	3.00 gm.	(gr. xivij).
Zinc. oxid.	. . . . .	8.00 gm.	(3lj).
Glycerin.	. . . . .	9.25 c.c.	(3iiss).
Aq. calcis	. . . . . q. s. ad	120.00 c.c.	(3iv).

After a few days' use of this lotion all manifestations will have subsided. Should the slight itching be still present or have resumed—no attention need be paid to itching during the day from a diagnostic point of view—a second course may be instituted, but this is scarcely ever necessary, except in very pronounced and long-standing cases.





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